This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

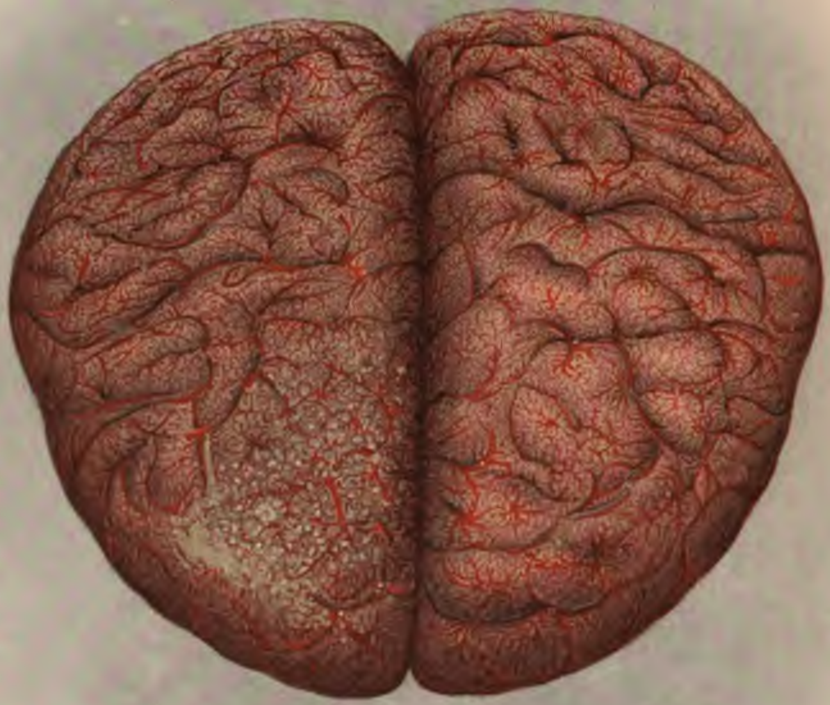
LANE MEDICAL LIBRARY STANFORD
P45 .R842 1901 STOR
Pediatrics : the hygienic and medical tr
24503435283

H. H. Rumsey

LAURENCE WELTI

LANE
MEDICAL LIBRARY
SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
1885
Gift
Dr. Lawrence Welti

PLATE I.



Tubercular Meningitis.

(Page 426.)

PEDIATRICS

[illegible]
[illegible]

CHILDREN

THOMAS MORGAN ROTCH, M.D.
[illegible]

[illegible]
[illegible]

[illegible]

[illegible]
[illegible]

PEDIATRICS

THE HYGIENIC AND MEDICAL TREATMENT
OF
CHILDREN

BY THOMAS MORGAN ROTCH, M.D.
PROFESSOR OF THE DISEASES OF CHILDREN, HARVARD UNIVERSITY

*THIRD EDITION, REARRANGED
AND REWRITTEN*

ILLUSTRATED BY NUMEROUS ENGRAVINGS IN THE
TEXT AND BY COLORED PLATES

PHILADELPHIA AND LONDON
J. B. LIPPINCOTT COMPANY
1901

Copyright, 1895, by J. B. Lippincott Company.

Copyright, 1901, by J. B. Lippincott Company.

ELECTROTYPED AND PRINTED BY J. B. LIPPINCOTT COMPANY, PHILADELPHIA, U. S. A.

TO

ABRAHAM JACOBI, M.D., LL.D.,

PROFESSOR OF DISEASES OF CHILDREN IN COLUMBIA UNIVERSITY,
NEW YORK,

THE MOST DISTINGUISHED

STUDENT AND TEACHER OF PEDIATRICS

IN AMERICA.

66 130

PREFACE.

WHEN another edition of this work was called for it was found necessary to rewrite it in order to bring it into accord with the advances which have been made in the subject of Pediatrics during the past six years. It is, therefore, offered to the Profession as practically a new book. The order in which the different subjects have been treated, and the relative space assigned to them, have in many instances been radically changed. The endeavor has been made to emphasize the practical character of the work by thoroughly systematizing the etiology, the symptomatology, the diagnosis, and the treatment of the various diseases. Much attention has been devoted to the anatomy and physiology of early life and to the advances which have been made in the subjects of infant feeding, of bacteriology, and of the blood. Several new colored plates and a number of radiographs have been added to the illustrations.

In acknowledging the aid which I have received I owe special recognition to my assistant and friend, Dr. Maynard Ladd, for his help in the preparation of the entire book. His interest and enthusiasm have been unflagging and invaluable. My thanks are also offered to those who have by their advice enabled me to remodel the book, and I would particularly acknowledge the information connected with the pathology which has been given so freely by Professor William T. Councilman. I am also under much obligation to Dr. John H. McCollum, Dr. Robert W. Lovett, Dr. John L. Morse, Dr. Algernon Coolidge, Jr., Dr. John Dane, and Dr. John T. Bowen. Dr. William P. Northrup supplied for the previous edition a number of valuable plates illustrating gastro-enteric diseases, and these plates are now retained. Dr. Franklin W. White has been of great service in the preparation of the article on the blood. Dr. Ernest A. Codman kindly furnished the plates of the radiographs.

The publishers have shown unfailing courtesy and much liberality.

Finally, I am greatly indebted to my medical associates throughout the country for their encouragement, which has both stimulated me in my work and greatly aided me in its completion.

T. M. ROTCH.

197 COMMONWEALTH AVENUE, BOSTON, MASSACHUSETTS.

CONTENTS.

DIVISION I.

THE NORMAL INFANT.

PAGE
I.—Introduction 2
II.—The Fœtal Circulation 19
III.—The Infant at Term 21–39
Vernix Caseosa; Skin; Cord; Spine; Neck; Head; Thorax; Abdomen; Bladder; Pelvis; Uterus; Temperature; Pulse; Respiration; Height; Weight; Vitality; Hands; Feet; Bone Marrow; Functions; Blood; Lymphatic System; Urine; Intestinal Discharges.

IV.—Normal Development 39–111
Cord; Spine; Neck; Head; Thorax; Abdomen; Temperature; Pulse; Respiration; Height; Weight; Feet; Bone Marrow; Skin; Functions; Lymphatic System; Thyroid Body; Urine; Intestinal Discharges; Infantile Skeletons; Normal Infants; Topographical Anatomy of Early Life; Nursery; Bathing; Clothing; Feet; Shoes; Sleep; When to go out; Nursery-Maids; Mouth; School; Defects of Posture.

DIVISION II.

FEEDING.

General Principles 112
Mammary Gland 114
First Nutritive Period 116–241
I.—Maternal Feeding 116
II.—Direct Substitute Feeding 160
Wet-Nurses; Animals.
III.—Indirect Substitute Feeding 164
Cows; Chemistry of Cow's Milk; Bacteriology of Cow's Milk; Milk-Laboratories and Percentage Feeding; Home Modification; Oat-Jelly; Barley-Water; Wheat; Artificial Foods; Matzoon; Kumyss; Peptonized Milk; Malted Foods.
Second Nutritive Period 241
Third Nutritive Period 245

DIVISION III.

GENERAL PRINCIPLES OF EXAMINATION AND TREATMENT.

Method of examining a Child 246–256
Lumbar Puncture; Röntgen Light; Treatment; Prophylaxis.

DIVISION IV.

PREMATURE INFANTS.

Determination of Age 257
Normal Development 259
Appearance at Birth 264
Weight 265
Incubator (Brooder) 266
Food 273

DIVISION V.

DISEASES OF THE NEW-BORN.

PAGE

Inheritance. Malformation. Traumatism 282

Maternal Impressions 285

Diseases of the Head 286–295

Caput Succedaneum; Cephalhæmatoma; Meningocele; Encephalocele; Anencephalia; Congenital Hydrocephalus; Harelip; Cleft Palate; Tongue-Tie; Ranula; Protrusion of Ears; Ophthalmia Neonatorum.

Diseases of the Neck 295–296

Hæmatoma of the Sterno-cleido-mastoid Muscle; Branchial Fistulæ.

Diseases of the Trunk 296–310

Mastitis; Depressed Sternum; Prominent Sternum; Spina Bifida; Rhachischisis; Phlebitis and Arteritis Umbilicalis; Congenital Umbilical Hernia into the Cord; Fungus of the Umbilicus; Meckel's Diverticulum; Umbilical Hernia; Inguinal Hernia; Femoral Hernia; Hydrocele; Undescended Testicle; Tumors of the Testis; Malformations about the Rectum; Occlusion of the Vagina; Hypospadias; Epispadias; Congenital Obliteration of the Intestine; Congenital Malformations of the Œsophagus and Stomach.

Diseases of the Extremities 310–312

Fingers; Toes; Club-Hand and Foot; Congenital Dislocation of the Hip; Congenital Dislocation of the Knee; Birth (Obstetrical) Paralysis.

General Diseases 312–325

Asphyxia; Acute Fatty Degeneration of the New-Born (Buhl's Disease); Infectious Hæmoglobinæmia of the New-Born; Hemorrhage in Early Life; Sclerema Neonatorum; Œdema Neonatorum; Icterus Neonatorum; Erythema Neonatorum.

DIVISION VI.

DISEASES OF NUTRITION.

Rhachitis (Rickets) 326–343

Congenital or Fœtal Rhachitis.

Osteomalacia 343

Scorbutus (Scurvy) 344

Infantile Atrophy 348

DIVISION VII.

DISEASES OF THE SKIN.

Scabies 355

Pediculosis 357

Impetigo Contagiosa 357

Furunculosis 358

Molluscum Contagiosum 358

Seborrhœa Capitis of Infants 359

Tinea Tricophytina 359

Tinea Favosa 360

Tinea Versicolor 360

Alopecia Areata 361

Pemphigus Neonatorum 361

Pemphigus 361

Epidemic Pemphigus Infantilis 362

Dermititis Exfoliativa Neonatorum (Ritter's Disease) 362

Dermatitis 363

Sudamina 365

Erythema 366

Erythema Intertrigo 367

PAGE
Erythema Nodosum 367
Erythema Urticatum (Urticaria) 367
Eczema 369
Psoriasis 372
Prurigo 373
Herpes Zoster 374
Pityriasis 375
Verrucæ (Warts) 375
Lentigo 376
Melanoderma Lenticularis Progressiva 376
Lichen 377
Ichthyosis 377
Scleroderma 379
Acute Circumscribed Œdema (Angio-Neurotic Œdema) 379

DIVISION VIII.

SPECIFIC INFECTIOUS DISEASES.

Tuberculosis 381–432
Acute Miliary Tuberculosis; Chronic General Tuberculosis; Tuberculosis of Lymph-Nodes, of Larynx, of Trachea, of Lungs, of Pleura, of Gastro-Enteric Tract, of Peritoneum, of Brain; Tubercular Meningitis; Tubercular Dactylitis; Tuberculosis of Thyroid Gland, of Thymus Gland, of Pancreas, of Spleen, of Liver, of Kidney, of Bladder, of Testicle, of Skin.
Epidemic Cerebro-Spinal Meningitis 432
Typhoid Fever 447
Diphtheria 459–476
Influenza (La Grippe) 476
Malaria 481
Erysipelas 497
Amœbic Ileo-Colitis 501
Cholera Infantum 502
Cholera Asiatica 505
Pertussis 505
Acute Infectious Osteomyelitis 511
Rheumatic Fever (Acute Articular Rheumatism) 514
Syphilis 520–542
The Exanthemata 542–613
Scarlet Fever; Measles; Rubella; Variola; Vaccinia; Varicella.
Parotitis (Mumps) 613

DIVISION IX.

DISEASES OF THE MOUTH, NOSE, EAR, NASO-PHARYNX, AND PHARYNX.

Diseases of the Mouth 615–640
Nomenclature; Stomatitis Catarrhalis; Stomatitis Herpetica; Stomatitis Ulcerosa; Stomatitis Mycetogenetica; Stomatitis Hyphomycetica (Thrush); Stomatitis Pseudo-Membranosa; Stomatitis Gangrænosa (Noma; Cancrum Oris) Glossitis; Lingua Geographica; Microglossia; Macroglossia; Difficult Dentition.
Diseases of the Nose 640–644
Rhinitis; Mucous Polypus; Epistaxis.
Diseases of the Ear 644
Diseases of the Naso-Pharynx 645–649
Hypertrophy of the Pharyngeal Tonsil (Adenoid Growths).
Diseases of the Pharynx 649–659
Tonsillitis; Peritonsillar Abscess; Pharyngitis; Elongation of Uvula; Retropharyngeal Abscess.

DIVISION X.

DISEASES OF THE LARYNX, TRACHEA, BRONCHI, LUNGS, AND PLEURA.

PAGE
DISEASES OF THE LARYNX 660–665
Laryngospasmus; New Growths; Foreign Bodies; Œdema; Laryngitis.
DISEASES OF THE TRACHEA 665
DISEASES OF THE BRONCHI AND LUNGS 665–707
Acute Bronchitis; Chronic Bronchitis; Fibrinous Bronchitis; Bronchial Asthma; Pneumonia; Pneumococcus Lobar Pneumonia; Lobar Pneumonia due to other Causes; Acute Broncho-Pneumonia; Chronic Broncho-Pneumonia; Hypostatic Pneumonia; Atelectasis; Emphysema; Gangrene and Abscess of the Lung.
DISEASES OF THE PLEURA 707–718
Acute Pleurisy, Dry, Sero-Fibrinous, Purulent (Empyema); Chronic Pleurisy; Hydrothorax; Pneumothorax.

DIVISION XI.

DISEASES OF THE HEART AND PERICARDIUM.

DISEASES OF THE HEART 719–753
Congenital Diseases 720–730
Open Foramen Ovale; Defect of the Ventricular Septum; Lesions of the Pulmonary Orifice; Persistence of the Ductus Arteriosus; Transposition of the Large Arteries; Lesions of the Tricuspid Orifice; Lesions of the Mitral and Aortic Orifices.
Acquired Diseases 730–748
Cardiac Hypertrophy; Cardiac Dilatation; Myocarditis; Endocarditis, Acute, Chronic; Mitral Insufficiency; Mitral Stenosis; Aortic Insufficiency; Aortic Stenosis; Tricuspid Insufficiency; Tricuspid Stenosis; Pulmonary Insufficiency and Stenosis.
Functional Diseases 748
DISEASES OF THE PERICARDIUM 754
Acute Pericarditis; Chronic Adhesive Pericarditis.

DIVISION XII.

DISEASES OF THE ŒSOPHAGUS, STOMACH, AND INTESTINE.

THE ŒSOPHAGUS 767
THE STOMACH 769–783
Developmental 774
Functional 774
Acute Nervous Vomiting 774
Cyclic or Persistent Vomiting; Gastralgia; Acute Gastric Indigestion (Acute Dyspepsia); Chronic Gastric Indigestion (Chronic Dyspepsia); Eliminative.
Organic 783
Contraction; Dilatation; Ulcers; New Growths; Acute Gastritis; Chronic Gastritis.
THE INTESTINE 793
Developmental 795
Functional 795
Acute Nervous; Acute Indigestion; Chronic Nervous; Tubular; Chronic Duodenal Indigestion; Chronic Intestinal Indigestion; Incontinence of Fæces; Constipation; Eliminative.
Organic 805
Acute Fermental; Chronic Fermental; Dilatation of Colon; Volvulus; Intussusception; Hernia; Fissures; Prolapse; Polypi; Hemorrhoids; Fistulæ; New Growths; Proctitis; Appendicitis; Ileo-Colitis; Animal Parasites.

DIVISION XIII.

DISEASES OF THE LIVER, PANCREAS, SPLEEN, AND PERITONEUM.

PAGE
THE LIVER 824
Icterus; Acute Yellow Atrophy; Congestion; Fatty Infiltration; Suppurative Hepatitis (Abscess); Hydatids; Biliary Calculi; New Growths; Amyloid; Interstitial Hepatitis (Cirrhosis).
THE PANCREAS 840
THE SPLEEN 840
THE PERITONEUM 840
Acute Peritonitis; Peritonitis of the New-Born; Acute Pneumococcus Peritonitis; Chronic Peritonitis.

DIVISION XIV.

DISEASES OF THE KIDNEYS, BLADDER, AND GENITAL ORGANS.

THE KIDNEY 844
Congenital; Acquired; Anuria; Physiological Albuminuria; Albuminuria of Adolescence: Hæmaturia and Hæmoglobinuria; Chyluria; Glycosuria; Active Hyperæmia; Passive Hyperæmia; Acute Diffuse Nephritis; Subacute Glomerular Nephritis; Chronic Interstitial Nephritis (Chronic Bright's Disease); Amyloid Infiltration; Acute Pyelitis and Pyelonephritis; Chronic Pyelitis; Perinephritis; Hydronephrosis; Malignant Growths and Enlargement.
THE BLADDER AND GENITALS 867
Acute Cystitis; Chronic Cystitis; Vulvo-Vaginitis; Orchitis; Epididymitis; Tumors of the Testicle; Phimosis; Enuresis (Incontinence); Masturbation.

DIVISION XV.

THE BLOOD. THE LYMPH-NODES. THE DUCTLESS GLANDS.

THE BLOOD 874–897
Nomenclature 874
Normal Conditions 875
Abnormal Conditions 879
Leukæmia 882
Pseudo-Leukæmic Anæmia of Infancy 887
Primary Anæmias 890
Pernicious Anæmia 890
Chlorosis 892
Secondary Anæmias 894
THE LYMPH-NODES 897–898
Simple Acute Adenitis 897
Simple Chronic Adenitis 898
THE DUCTLESS GLANDS 899–909
Diseases of the Thyroid Gland 899
Goitre (Bronchocele) 899
Myxœdema; Cretinism 901
Exophthalmic Goitre 905
Acute Thyroiditis 907
Tumors of the Thyroid Gland 907
Diseases of the Thymus Gland 907
Diseases of the Adrenal Glands 908
Addison's Disease 908

DIVISION XVI.

DISEASES OF THE NERVOUS SYSTEM.

PAGE
CONVULSIONS 910
CHOREA 911
EPILEPSY 916
INSANITY 925
IDIOCY 930
MICROCEPHALUS 932
MIRROR WRITING 932
HYSTERIA 932
HYPNOTIC STATE 935
CATALEPSY 935
SIMULATED DISEASES 935
INSOLATION 935
CONCUSSION 937
TEMPORARY AMNESIA 937
TEMPORARY APHASIA 937
ARRESTED PSYCHICAL DEVELOPMENT 938
RETARDED SPEECH 938
HEADACHES 939
VERTIGO 940
PAVOR NOCTURNUS (NIGHT-TERRORS) 941
TREMOR 942
TETANY 942
DENTAL REFLEXES 943
NYSTAGMUS 944
GYROSPASM AND SPASMUS NUTANS 944
REFLEX SYMPTOMS OF THE EAR 944
REFLEX SYMPTOMS OF THE LARYNX 944
PAROXYSMAL GASPING 946
REFLEX SYMPTOMS OF THE LUNG 946
REFLEX COUGH 947
REFLEX SYMPTOMS OF THE HEART 948
REFLEX SYMPTOMS OF THE STOMACH 948
REFLEX SYMPTOMS OF THE BLADDER 948
REFLEX SYMPTOMS OF THE VAGINA 948
REFLEX SYMPTOMS OF THE RECTUM 949
CEREBRAL ABSCESS 949
CEREBRAL PARALYSIS (INFANTILE CEREBRAL PALSIES) 950
MYELITIS 958
INFANTILE SPINAL PARALYSIS (POLIOMYELITIS ANTERIOR ACUTA) 958
PARALYSIS CAUSED BY CARIES OF THE SPINE 967
OBSTETRICAL PARALYSIS 968
NEURALGIA 970
EPIPHYSEAL HYPERÆMIA (GROWING PAINS) 970
HYDROCEPHALUS 970
THROMBOSIS OF THE CEREBRAL SINUSES 975
ATHETOSIS 976
INTRA-CRANIAL TUMORS 978
CEREBRAL SYPHILIS 980
MENINGITIS 981
ACUTE ENCEPHALITIS 984
BULBAR PARALYSIS 985
MULTIPLE NEURITIS 985
INSULAR OR DISSEMINATED SCLEROSIS 987
HEREDITARY ATAXIA (FRIEDRICH'S DISEASE) 988
LOCOMOTOR ATAXIA 989

PAGE
Syringomyelia 989
Hereditary Spastic Paralysis 989
Progressive Central Muscular Atrophy 989
Progressive Neural Muscular Atrophy 991
Progressive Muscular Dystrophies 993

DIVISION XVII.

UNCLASSIFIED DISEASES.

Hæmophilia 997
Purpura 998
Status Lymphaticus (Lymphatism) 1000
Muscular Rheumatism (Myalgia) 1001
Arthritis Deformans 1003
Chronic Rheumatism 1003
Diabetes Insipidus 1003
Diabetes Mellitus 1004

LIST OF TABLES.

TABLE PAGE

1. Relation of weight to vitality 37
2. Length of spine to sacrum 40
3. Relation of cricoid to sternum 43
4. Circumferences of head and thorax from birth to thirteen years 45
5. Proportions of face to cranium 45
6. Height of posterior nares 47
7. Temporary teeth. First dentition 50
8. Permanent teeth. Second dentition 51
9. Post-natal changes of fœtal conditions 55
10. Weights of the heart during its development 55
11. Amounts of food in an especial case 67
12. Three hundred and forty-one infants fed at the milk-laboratory 68
13. Temperature of infant at term 70
14. Townsend's temperature observations 71
15. Pulse-rate for males 71
16. Townsend's pulse observations 72
17. Respirations in infancy and childhood 72
18. Respirations in infants awake, asleep, and crying 72
19. Rate of growth in girls and boys and the relation between growth and disease 74
20. General figures of weight 79
21. Average height and weights from birth to five years 81
22. Average analysis of urine in infancy and childhood 86
23. Temperature of the bath for different ages 97
24. Average analysis of five specimens of human colostrum milk 123
25. General average of twenty-six analyses of human colostrum milk 123
26. Average analysis of human milk 127
27. Analyses of fore-milk, middle-milk, and strippings 133
28. Human breast-milk analyses 134
29. Intervals and number of day and night feedings 136
30. Analysis of typical normal, poor, over-rich, and bad milk 140
31. Analyses of human milk 143
32. Analyses of human milk 143
33. Analyses of human milk 144
34. Analyses of human milk 144
35. Analyses of human milk 144
36. Analyses of human milk 145
37. Analyses of human milk 145
38. Analyses of human milk 146
39. Alkalinity of cream mixtures corresponding to that of human milk 175
40. Cow's milk as compared with human milk 180
41. General rules for feeding during the first year 188
42. Practical limits of laboratory modification 199
43. Practical limits of laboratory modification 199
44. Table of whey-cream mixtures 201
45. Showing the management of the food and the increase in weight of a healthy infant during the first fifty-two weeks of its life 207

TABLE PAGE

46. Showing the management of the food and the increase in weight of a healthy infant during the first fifty-two weeks of its life 209
47. Showing lowest possible proteids with creams of different strengths 219
48. Showing lowest possible proteid mixtures of two, three, four, and four and one-half per cent. of fat made from ten per cent. cream 220
49. Showing lowest possible proteids in mixtures of two, three, four, and four and one-half per cent. of fat made from twelve per cent. creams 220
50. Showing variation of fat percentage in milk of different herds 221
51. For the calculation of home modifications 230
52. Showing fat percentages obtained by diluting twenty per cent. cream with whole milk 231
53. Showing dilutions of milk with sugar solutions 232
54. Showing dilutions of whole milk with sugar solution 232
55. Showing dilutions of eight per cent. cream with sugar solution 232
56. Showing dilution of twelve per cent. cream with sugar solution 232
57. Showing dilution of sixteen per cent. cream with sugar solution 233
58. Showing combination of fats and proteids obtained with creams of different strengths and whey 234
59. Weight for sixty-one days of infant premature at thirty-two weeks 276
60. Showing details of sixty-four days of life in the incubator of an infant premature at thirty weeks .... facing 281
61. Differential diagnosis of cerebral meningitis 415
62. Statistics of tuberculosis of the joints in the Children's Hospital 427
63. The doses of tincture of digitalis, strychnine, nitroglycerin, and atropine at different ages 470
64. The principal combinations of paroxysms caused by the plasmodium malariæ 486
65. One thousand cases of scarlet fever, by ages, with the deaths 545
66. Individuals living in houses invaded by variola 602
67. Differential diagnosis between varicella and variola 609
68. Differential diagnosis of the exanthemata 612
69. Classification of the diseases of the mouth 616
70. Amount of chlorate of potassium at different ages 624
71. Differential diagnosis between a dilated heart and a pericardial exudation 761
72. Classification of diseases of the gastric enteric tract 773
73. Normal average number of blood-corpuscles at different ages in cases in which there was a loss of weight in the first forty-eight hours 878
74. Percentages of leucocytes in the normal blood of infants and adults 879
75. Average percentages of the different elements of the blood in the splenic myelogenous and lymphatic leukæmias 884
76. Differential diagnosis between cerebral paralysis and poliomyelitis anterior 962

LIST OF ILLUSTRATIONS.

COLORED PLATES AND RADIOGRAPHS.

PLATE PAGE
I. Tubercular meningitis, convexity of the brain *Frontispiece.*
II. Icterus neonatorum. Red bone-marrow. Yellow bone-marrow. Erythema neonatorum 80
III. Intertrigo. Seborrhœa capitis. Umbilical cords. Napkins 84
IV. Radiograph of the chest and abdomen 254
V. Radiographs of the femora, tibiæ, and fibulæ in rhachitis, and of the tibiæ and fibulæ in syphilis 336
VI. Tubercular meningitis, base of the brain 424
VII. Radiographs of osteomyelitis of the lower end of the femur, and of tuberculosis of the knee-joint 512
VIII. Scarlet fever. Measles 564
IX. Vaccination 604
X. Varicella. Erysipelas. Syphilis 610
IX. Mouth and throat in thrush, varicella, stomatitis herpetica, stomatitis ulcerosa, follicular tonsillitis, diphtheria 620
XII. Blood-corpuscles. Mosquitoes, genus anopheles; genus culex 874

FIGURES.

FIG.
1. Fœtal circulation 19
2. Heart, natural size, at two days 20
3. Spinal curves 23
4. Skull of infant at term, natural size 25
5. Section of fœtal lung at five months 32
6. Section of infant's lung at ten months 32
7. Lobulated kidney, natural size 34
8. Stomach, natural size. Infant, three hours old 34
9 Respiration at birth, illustrative diagram 36
10. Relative circumference of head, thorax, and abdomen 44
11. Infant skull, natural size 46
12. Skulls showing development of ramus at birth, and at three years 49
13. Five periods of development in the first dentition 50
14. Eight periods of development in the second dentition 52
15. Respirations for one-half minute in a healthy infant nine months old 53
16. Frozen section, child of three years 54
17. Heart, showing Eustachian valve and foramen ovale 55
18. Heart, showing ductus arteriosus 55
19. Stomach, spleen, and pancreas at ten months 57
20. I., II., III., and IV. Gastric capacity in the first five months of life 60
21. Stomach of infant two and one-half days old, natural size 64
22. Stomach of infant five days old, natural size 64
23. Stomach of infant seven days old, natural size 65
24. Stomach of infant twelve days old, distended to hold eighty c.c. 65
25. Stomach of infant five months old, distended to hold two hundred and twenty-five c.c. 66

FIG PAGE
26 Stomach of infant seven months old, natural size 66
27. Stomach of infant nineteen months old, natural size 67
28. Skeleton of infant at term 87
29. Skeleton of infant at nineteen months 87
30 and 31. Normal infant seven months old 89
32 and 33. Normal development at six years 90
34 and 35. Normal development at twelve years 92
36. Infant's bed, Infants' Hospital 94
37, A, B, C, D, E. Long clothes for infants 100
38, F, G, H, I, J. Short clothes for infants and children 103
39. Shape of soles for a child's shoe 105
40. Posterior spinal curvature from sitting too soon 110
41 and 42. Lateral curvature of the spine. Child, four and one-half years old 111
43. Breast-pump 120
44. Colostrum milk from cow (photomicrograph) 152
45. Colostrum milk from women (photomicrograph) 152
46. Stomach from infant five days' old and glass cylinder of same capacity 187
47. Centrifugal separator 191
48. Babcock fat-tester 192
49. Apparatus for the transportation of milk 193
50. Ice-box for the transportation of modified milk 194
51. Unmodified cow's milk 204
52. Cow's milk separated and recomposed 204
53. Human milk 205
54. Modified cow's milk 205
55. Apparatus for home modification 224
56. Sugar-measure 225
57. Jar containing milk, cream, and siphon 226
58. Phonendoscope 251
59. Tongue depressor 251
60. Fœtal stomach, natural size, four and one-half months old 260
61. Fœtal stomach, natural size, seven and one-half months old 260
62. Fœtal stomach, natural size, eight months old 260
63. Fœtal stomach, natural size, eight months old 261
64. Infant premature at seventh month 264
65. Infant premature at the twenty-eighth week 267
66. Incubator for premature infants 268
67. Section of incubator 270
68. Feeder for premature infants 272
69. Infant premature at thirty weeks. Age, nine months 278
70. Caput succedaneum. Male, two hours old 286
71. Double cephalhæmatoma. Infant, four days old 287
72. Double external cephalhæmatoma. Both parietal bones 288
73. External cephalhæmatoma. Parietal bone dissected 288
74. Meningocele. Female, three years old 289
75. Hydro-encephalocele. Female, two months old 291
76. Double harelip 292
77. Congenital depression of sternum. Male, six years old 297
78. Spina bifida. Male, four and one-half years old 299
79. Spina bifida of dorsal lumbar region 300
80. Spina bifida of lumbar region 301
81. Large umbilical hernia. Infant, five months old 303
82. I. Normal bone. II. Bone of cretin. III. Rhachitic bone 329
83. Spindle-shaped rhachitic bone 331
84. Inner surface of sternum, showing rhachitic rosary 335
85. Rhachitic kyphosis 335
86. Rhachitis, with enlarged spleen. Male, three years old 337
87, I. and II. Rhachitis. Age, six years 342

FIG. PAGE
88. Congenital rhachitis 343
89. Vertical section of a leg in a case of infantile scorbutus 345
90. Section of scorbutic bone 345
91. Infantile scorbutus. Female, ten months old 348
92. Infantile atrophy. Female, nine months old 352
93. Infantile atrophy. Female, ten months old 352
94. Infantile atrophy. Female, one and one-half years old 353
95. Infantile atrophy, showing extreme emaciation 354
96. Tinea tonsurans. Male, eight years old 360
97. Eczema capitis 371
98. Method of treating eczema capitis 371
99. Chronic pulmonary tuberculosis with involvement of the cervical lymph-nodes. 391
100. Chronic tuberculosis of the lungs. Female, eight years old 398
101. Tubercular ulcers of the colon 400
102, I. Tubercular ulcers of the small intestines 400
102, II. Large tubercular ulcer of the cæcum 401
103. Tubercular peritonitis. Male, nine years old 404
104. Tubercular peritonitis. Male, four years old 405
105. Tubercular meningitis. Male, three years old 418
106. Recurrent tubercular meningitis. Female, twenty-one months old 422
107. Tubercular dactylitis 430
108. Chronic intermittent cerebro-spinal meningitis. Female, eight years old 443
109. Secondary choroido-iritis, occurring in cerebro-spinal meningitis 444
110. Chronic cerebro-spinal meningitis, with spastic condition of the hand 445
111. Typhoid fever. Male, five years old 457
112. Typhoidal ileo-colitis, showing ulcers of the colon. Female, two years old 458
113. Enlarged Peyer's patches closely simulating the lesions of ileo-colitis 459
114. Irrigation of the nose in diphtheria 473
115. Malaria. Enlarged spleen. Boy, nine years old 491
116. Malaria. Enlarged spleen. Girl, nine years old 493
117. Pertussis during paroxysm. Female, four years old 508
118. Acute articular rheumatism. Adult type of the disease. Male, three and one-half years old 518
119. Rheumatism. Subcutaneous fibrous nodules. Male, thirteen years old 519
120. Syphilitic maculæ, ulcers, and bullæ on the soles of the feet. Male, two and one-half months old 535
121. Congenital syphilis. Enlarged spleen. Male, two and one-half months old 536
122. Syphilitic teeth of the second dentition 537
123. Late manifestations of syphilis. Female, three and one-half years old 540
124. Hereditary syphilis. Male, six years old. Abnormal prominence of frontal bone 541
125. Interstitial nephritis. Section of kidney from child with scarlet fever 551
126. Capsular glomerulo-nephritis. Section of kidney from child with scarlet fever 552
127. Typical condition of the face in measles, fifteen days from infection. Female, six years old 587
128. Varicella. Stage of efflorescence, third day 610
129. Enlargement of submaxillary glands 614
130. Mycelium of thrush, interspersed with spores and fatty degenerated cells 628
131. Thrush, showing the formation of spores in the mycelium 629
132. Stomatitis gangrænosa, left cheek. Female, four years old 632
133. Stomatitis gangrænosa, secondary to measles and pneumonia. Female, five years old 632
134. Showing the connection between the sensori-motor nerves and the sympathetic 635
135. Showing the anatomical nervous connection between the teeth and the membrana tympani 636
136 and 137. Showing the relations of the teeth to the gums in difficult dentition 637
138. Gum-lancet 638
139. Hypertrophy of the pharyngeal tonsil (adenoid growths). Female, ten years old 648
140. Retropharyngeal abscess. Male, seven months old 658

FIG. PAGE
141. Retropharyngeal abscess secondary to cervical spondylitis 659
142. Section of a child's lungs in acute bronchitis 667
143. Lobar pneumonia. Female, eight years old 688
144. Lobar pneumonia. Three invasions. Male, six years old 688
145. Broncho-pneumonia complicating measles. Early stage 692
146. Bronch-pneumonia secondary to diphtheria 693
147. Chronic broncho-pneumonia 695
148. Acute broncho-pneumonia. Female, four and one-half years old 702
149. Congenital cardiac disease. Unclosed ventricular septum. Female, ten months old 727
150. Congenital cardiac disease. Stenosis of pulmonary orifice. Incomplete septum ventriculorum. Male, four and one-half years old 728
151. Congenital cardiac disease. Transverse section of heart near apex, showing thickened septum ventriculorum 728
152. Congenital cardiac disease. Open ductus arteriosus. Male, sixteen days old 729
153. Acute endocarditis. Mitral insufficiency. Lacking compensation. Orthopnœa. Female, nine years old 750
154. Chronic recurrent endocarditis. Mitral insufficiency. Disturbance of compensation. Dilated heart. Enlarged liver. Œdema of lungs. Ascites. Male, ten years old 752
155. Chronic endocarditis. Mitral insufficiency. General œdema and anasarca 752
156. Chronic endocarditis. Mitral insufficiency. Returning compensation. Enlarged liver. Enlarged heart 753
157. Chronic endocarditis. Mitral insufficiency with returned and complete compensation 753
158. Areas of absolute dulness in enlarged heart and in distended pericardium 760
159. Congenital dilatation of the œsophagus. Female, ten weeks old 768
160. Colon showing presence of bismuth which had been given by the mouth 774
161. Dilated stomach. Rhachitic infant, seven months old 784
162. Dilatation of the stomach. Age, six years 787
163. Follicular ulceration of the stomach. Female, one year old 788
164. Dilatation of the colon. Male, twelve years old 810
165. Hyperplasia of the lymph-follicles 826
166. Non-ulcerative follicular inflammation. Simple hyperplasia of the lymph-follicles. Female, three years old 826
167. Colitis follicularis non-ulcerativa. Male, two years old following 826
168. Colitis follicularis non-ulcerativa " 826
169. Microscopic section of hyperplasia of lymph-follicles (solitary glands). " 826
170. Microscopic section of follicular ulceration of the colon " 826
171. Ileo-colitis. Ulcerative follicularis. Infant, sixteen months old " 826
172. Acute ulcerative catarrhal colitis. Female, three months old " 826
173. Inflammation of follicles and surrounding parts of the colon, with necrosis. Female, three months old " 826
174. Pigmented follicular ulcers of the colon. Chronic catarrhal ulcerative follicular colitis " 826
175. Pseudo-membranous colitis. Child, three and a half years old " 826
176. Microscopic section. Pseudo-membranous colitis. Female, four years old 827
177. Microscopic section. Superficial necrosis of the mucosa of the colon, with swelling of the lymph-follicles 827
178. Oxyuris vermicularis. Ascaris lumbricoides 830
179. Tæniæ, I., without head; II., with head 833
180. Amyloid liver. Pulmonary tuberculosis. Male, seven and three-quarter years old 838
181. Hypertrophic cirrhosis. Female, eighteen months old 839
182. Acute diffuse nephritis, following scarlet fever 855
183. Probable chronic parenchymatous nephritis with an acute exacerbation. Male, eleven years old 860
184. Probable chronic parenchymatous nephritis with an acute exacerbation. Female, nine years old 861

FIG. PAGE
185. Chronic cervical adenitis.. 899
186. Hyperæmia of the thyroid gland. Female, thirteen years old................ 900
187. Myxœdema. Female, five and one-half years old 903
188. Myxœdema. Female, nine years old.................................... 904
189. Rheumatic arthritis. Endocarditis. Cardiac enlargement. Chorea. Female, eight years old 922
190. Reflex connection between the ear and larnyx.............................. 946
191. Cerebral paralysis. Spastic paraplegia. Cross-legged progression. Male, five and one-half years old 957
192. Cerebral paralysis. Diplegia. Female, five years old....................... 957
193. Poliomyelitis anterior. Left leg. Female, nine years old 965
194. Poliomyelitis anterior. Abdominal muscles, left side. Female, two and one-half years old.. 965
195. Poliomyelitis anterior. Flail-leg, left side. Male, six and one-half years old.. 966
196. Poliomyelitis anterior. Talipes equinus, right side. Male, eleven and one-half years old 966
197. Hydrocephalic brain.. 972
198. Hydrocephalic skull, child three years 972
199. Normal skull, child three years 972
200. Congenital internal hydrocephalus. Male, seven months old 974
201. Congenital internal hydrocephalus. Female, five years old.................. 974
202. Congenital athetosis. Female, two years old................................ 977
203. Pseudo-hypertrophic muscular paralysis, showing enlarged calves............. 995
204. Pseudo-hypertrophic muscular paralysis, showing position assumed in rising from the floor.. 995
205, I. and II. Acute rheumatic torticollis. Fifth day of attack................. 1002

PEDIATRICS.

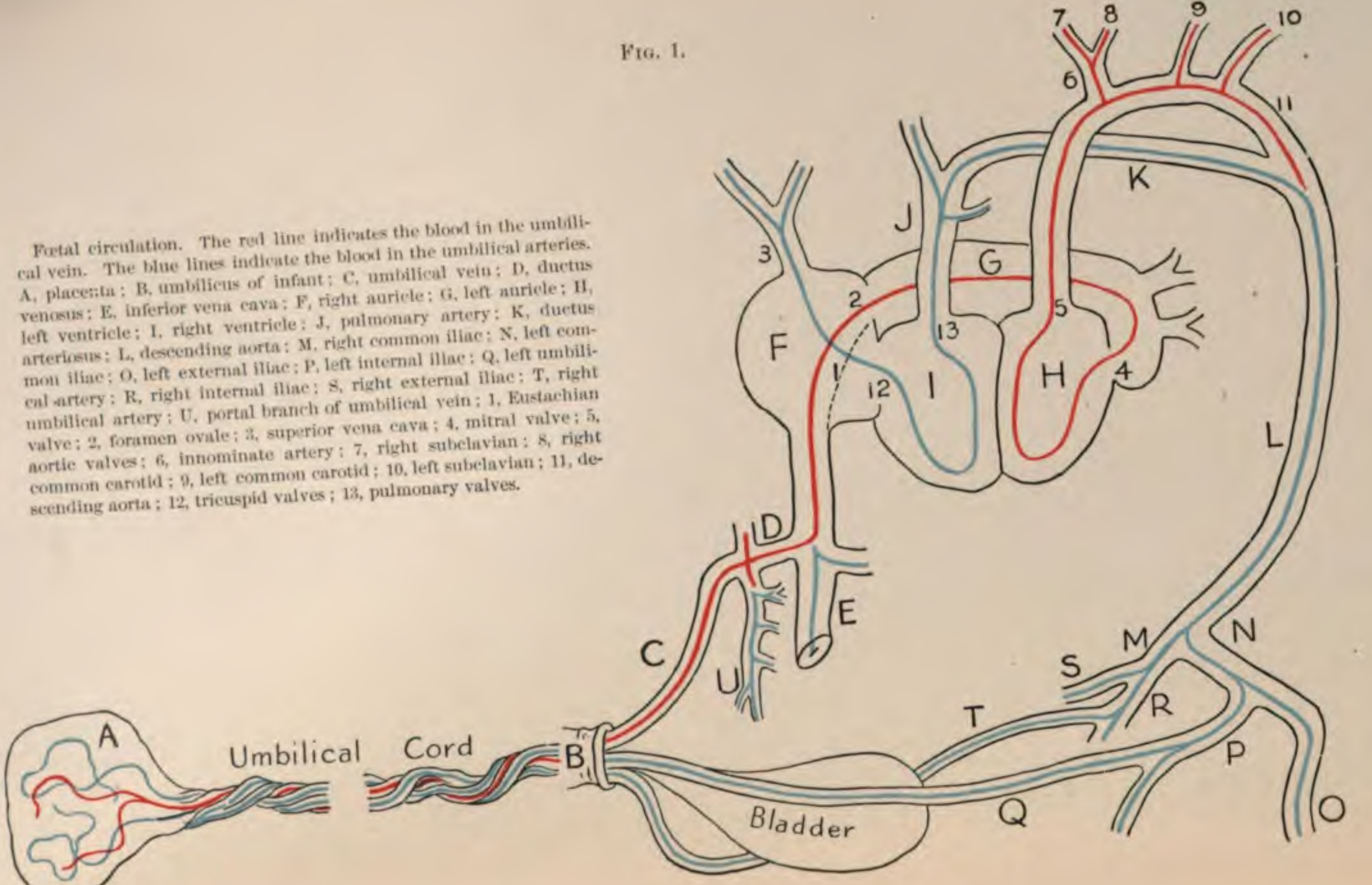
DIVISION I.

THE NORMAL INFANT.

I. INTRODUCTION.

PEDIATRICS is a branch of medicine of the greatest practical importance. Those who enter into general practice will at once be called upon to treat infants and children. The proper appreciation of the sensitive temperaments and needs of this class of patients will be of great aid in successfully establishing a practice among those whose favorable opinion may make or mar professional success. The difficulties to be surmounted in correctly diagnosticating and treating young children are far greater than those which are encountered in adult life. The reason for this is that for adult cases there is some standard by which we can be guided, being ourselves adults. What standard, however, is there for the feelings and sensitive organization of the child? We have none within ourselves; it must all come from long and patient observation, with its resulting experience. The mere knowledge that certain diseases exist, and the usual methods of diagnosticating them, prove to be very inadequate when we are brought face to face with a sick and fretful child, or with an infant who is unable to describe its symptoms. Much additional knowledge is needed to enable us to understand the variety of symptoms which may arise in the same disease according to the age and individuality of the patient. It is now well recognized that there is a necessity for making a special study of children beyond what is learned in the general clinical study of adults. As our knowledge advances, we learn to appreciate that the various methods of treatment must be modified to correspond not so much to the special disease as to the special group of symptoms brought about by the age of the individual and the phase of its development. In studying, then, the different stages of development in children, we are in reality acquiring an alphabet, which when once thoroughly mastered will enable us to read the otherwise obscure language presented to us for translation by the various diseases of early life. The proper method of learning to understand sick infants and children is first to notice their peculiarities in health and to follow these peculiarities through the different stages of their development up to puberty. Thus, a pulse which would indicate an abnormal condition in the adult, or a convulsion which would be of serious import in the older subject, may often be but physiological or of slight consequence in the child. In fact, there are a large number of physiological and anatomical truths concerning the young the knowledge of which will simplify to a great degree otherwise almost insurmountable difficulties in diagnosis. The lack of this preliminary training, this alphabet, places the student who is endeavoring to understand diseases in children in the position of attempting to read without having first learned his letters. It is the province of pediatrics to begin with the human being at birth, to study it as it appears in the early hours of life, and to follow it in its development during the periods of infancy and childhood up to the age of puberty. For purposes of simplicity, we speak of infants and children, the anatomical and physiological conditions being sufficiently apparent to warrant this distinction between them. The period of infancy is usually spoken of as covering about the first two years of life. Its most distinctive features are presented in the first twelve months, the second year, month by month, rapidly approaching the conditions which exist in childhood. The second year, however, is influenced to such a degree by the various growing functions and tissues that its picture both in health and in disease resembles more closely the infant than the child. Childhood is empirically reckoned from the end of infancy to puberty, or the beginning of adolescence. A distinction must be made between the sexes, the girl becoming a fully developed woman some years before the boy becomes a man. The age of puberty is usually reckoned as beginning from the twelfth to the fourteenth year. Much latitude as to age, however, must be given for the special idiosyncrasy of the individual, and also for the climate, as it has been found that children who live in a warm climate arrive at the age of puberty much earlier than those who are exposed to the lower ranges of temperature. In taking the period of birth as a starting-point for our studies we must not overlook the fact that it is simply a stage of development with which we are dealing, and not a perfected being. The better, therefore, we understand the evolution of the embryo to the infant, the better shall we be prepared to appreciate the evolution of the infant to the child and of the child to the adult. It is especially important to understand the stage of development which exists just before birth, for on this depends the knowledge whether we have a physiologically and anatomically normal being before us, or one that is abnormal. Disease does not merely mean a pathological change in the tissues, but, as is especially well exemplified in the infant, may simply mean a retardation or arrest of development. Thus, what would be perfectly normal anatomically at the seventh month of intra-uterine life may at birth be abnormal, and hence constitute a

FIG. 1.



Fœtal circulation. The red line indicates the blood in the umbilical vein. The blue lines indicate the blood in the umbilical arteries. A, placenta; B, umbilicus of infant; C, umbilical vein; D, ductus venosus; E, inferior vena cava; F, right auricle; G, left auricle; H, left ventricle; I, right ventricle; J, pulmonary artery; K, ductus arteriosus; L, descending aorta; M, right common iliac; N, left common iliac; O, left external iliac; P, left internal iliac; Q, left umbilical artery; R, right internal iliac; S, right external iliac; T, right umbilical artery; U, portal branch of umbilical vein; 1, Eustachian valve; 2, foramen ovale; 3, superior vena cava; 4, mitral valve; 5, aortic valves; 6, innominate artery; 7, right subclavian; 8, right common carotid; 9, left common carotid; 10, left subclavian; 11, descending aorta; 12, tricuspid valves; 13, pulmonary valves.

disease. In like manner what may be normal at birth may if it persists into the second and third weeks become an abnormal condition. Disease, therefore, is a relative term. We may, however, simplify our classification of diseases by adopting two broad divisions corresponding to the changes which take place during intra- and extra-uterine life and designated as *congenital* and *acquired*. By congenital diseases we mean those resulting from changes occurring during intra-uterine life. These may arise from an arrest of development or from a continuation of normal intra-uterine conditions beyond the usual period of their cessation; also those which are caused by pathological processes such as inflammation. By acquired, we mean a pathological condition of existing tissues occurring after birth, and without regard to the stage of development.

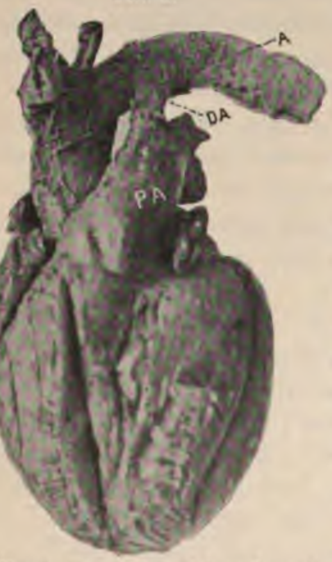
If we thoroughly understand the anatomical conditions existing just before birth, we can intelligently examine the young human being as it emerges from the uterus, and can judge in the early days of its existence whether we have under our care a normal infant or one that is to need special treatment.

II. THE FOETAL CIRCULATION.

The chief anatomical change which takes place at birth is the transition from the intra-uterine circulatory mechanism to a form adapted to extra-uterine life; in other words, from the oxygenation of the blood through the placenta to the same process carried on by the lungs. A general knowledge of the fœtal circulation is, then, evidently of considerable importance, especially when it is considered that a large proportion of the cases of congenital heart disease which we are called upon to diagnosticate is represented by perfectly normal prenatal conditions, such as absence of the ventricular septum, an open ductus arteriosus, or a patent foramen ovale.

Fig. 1 represents the course of the (red) oxygenated blood from the placenta to the infant, and that of the darker (blue) deoxidized blood from the infant back to the placenta. We must consider that in the fœtus the lungs are in a collapsed, inert condition, performing no part in the fœtal economy, but remaining quiescent until called upon to perform their special function at birth. The true lung of the fœtus, therefore, is represented by the placenta of the mother. It is here that the blood is oxygenated, and is carried by means of the umbilical vein directly through the umbilicus of the fœtus to the liver, as seen in the diagram. In the liver, the umbilical vein divides into three branches: (1) the smallest, carries the blood directly to the liver tissue, whence it is returned as in the adult to the inferior cava by the hepatic veins; (2) the largest portion meets and mixes with the blood from the portal system, and is distributed with it to the liver; (3) the remaining portion is carried, by a vessel called the *ductus venosus*, directly to the inferior cava, where it meets the deoxidized blood from the lower extremities, mixes with it, and is carried to the right auricle: here, instead of passing as in the adult into the right ventricle, it is directed by a membrane, called the *Eustachian valve*, through an opening between the two auricles, called the *foramen ovale*, into the left auricle. It then passes into the left ventricle through the mitral valve, and thence through the aortic valve into the aorta. The greater part of the blood-current is then carried by the carotid and subclavian arteries to the head and upper extremities, where, after doing its work in vitalizing the tissues and taking up their waste (a small portion also passing, as usual, into the descending aorta), it is returned as deoxidized blood through the veins to the superior cava into the right auricle, thence through the tricuspid valves into the right ventricle, and up through the pulmonary artery, where a small portion is distributed as usual to the lungs, while the remaining portion is carried directly over to the descending aorta by a vessel called the *ductus arteriosus*. It here mixes with the small portion of oxygenated aortic blood mentioned above, and passes down the aorta, being distributed on its way, as in the adult, until it reaches the internal iliac arteries. From these arteries it is carried, by branches called the *umbilical arteries*, through the umbilicus back to the cord and placenta. Thus, by simply referring to this diagram, we can tell at a glance which part of the young infant should be most developed, and the reasons for it. A noticeable point of clinical interest, in tracing the course of the fœtal circulation, is that the fresh oxygenated blood is mainly carried to the liver, head, and upper extremities, while the devitalized blood is distributed to the thorax and lower extremities. We should therefore expect, and we shall find it to be true, when we examine a normal new-born infant, that the head is larger than the thorax, that the abdomen is prominent from containing the large liver, and that the legs are insignificant and poorly developed.

FIG. 2.



Heart, natural size, at two days. A marks the aorta; PA marks the pulmonary artery; DA marks the ductus arteriosus.

When the placental circulation is cut off, an increased amount of blood is carried by the pulmonary artery to the lungs, and by degrees the fœtal circulation is replaced by that of extra-uterine life.

The *ductus venosus* and *ductus arteriosus* become fibrous cords.

The *Eustachian valve* disappears.

The *foramen ovale* closes.

The *umbilical vein* and *umbilical arteries* become obliterated, with the exception of the lower parts of the latter.

During the first two weeks of infancy we may have conditions existing physiologically which after that time would become pathological, and hence, to be well grounded in the diagnosis of disease in the infant, we must appreciate the importance of these facts.

Fig. 2 represents a heart taken from an infant two days old. It is of natural size, and shows the ductus arteriosus connecting the aortic and pulmonary arteries.

III. THE INFANT AT TERM.

By the infant at term we mean one that has been born at the termination of what is considered the usual period of pregnancy, two hundred and eighty days.

SKIN.—A normally developed fœtus when it first emerges from the uterus has a reddened skin and is covered thickly in many parts by a substance made up of the contents of the amniotic sac, in which the fœtus has been floating, and of the excretion of the sebaceous glands. This substance is called the *vernix caseosa*. In certain rare cases also this sebaceous matter is so universal and so impenetrable as to constitute a disease of serious import, and at times even to cause death. Infants also may be born with the skin almost entirely free from the vernix caseosa. After the infant has been bathed, and the vernix caseosa has been removed, it should present the color of a healthy skin reacting normally to its external surroundings. The skin should usually be some shade of delicate pink.

GENERAL DESCRIPTION.—The *body* and *limbs* should be well rounded, the *cry* vigorous, the extremities warm, and the grasp of the *hands* strong and active. The *hair* at birth is often thick, dark, and quite long, perhaps 2 to 5 cm. (1 to 2 inches), but we also frequently find the hair to be short, fine, some shade of light brown, small in amount, and the temples bald, the hair coming down to a round point on the forehead. The *eyes* are almost always half open when awake, expressionless, and of a dull grayish blue. The *head* is large in comparison with the *thorax*, the *arms* more rounded and large in proportion to the *legs*, and the *abdomen* is prominent.

The *cord* dries up and falls off at about the sixth or seventh day. It is important to know how it should look normally up to the day when it separates from the umbilicus in order to distinguish it from abnormal conditions. There is a slightly reddened areola where it joins and is to part from the abdominal wall. (Plate III.)

Palpation, percussion, and auscultation show that the *heart* has about the same proportionate position in reference to the *lungs* as is found in the adult, but that the *liver* occupies much more space, coming fully 1 to 2 cm. (½ to 1 inch) below the edge of the ribs in the right hypochondriac and the epigastric regions, and encroaching on the lung-space in the right back to the extent of fully one rib and interspace. The *testicles* have descended, and the *bladder*, when full of urine, presents an area of dulness of about 2 cm. (1 inch) just above the pubis in the median line. It is an important fact that the bladder is an abdominal rather than a pelvic organ in the infant and young child. The dull area of the *spleen* corresponds in its position to that found in the adult, but is scarcely perceptible.

The following anatomical and physiological truths relating to the infant at term are based on what is usually found to exist in the average infant. I am especially indebted to Professor Thomas Dwight for the assistance which he has given to me from his own original investigations, and from his verification of the anatomical part of the work. It has not been attempted to give the complete anatomy and physiology of the various periods of early life, but merely to pick out the practical points in these periods which will aid in clinical diagnosis and treatment. The great importance of thoroughly understanding the normal anatomy and physiology of human beings before attempting to deal with the morbid conditions which arise in them is now so well recognized that no preliminary remarks are needed to show how vital to all advance in clinical medicine is the proper reading of anatomical and physiological truths. There are several points in the anatomy and physiology of the new-born infant which would be better understood if the fact were borne in mind that in many respects the body at this age is more adapted to its intra-uterine life and to its means of exit into the external world than to the conditions which surround it in extra-uterine life.

It is not uncommon at birth for the face to be swollen and the features out of shape; this comes from pressure, and will soon pass away.

At birth the trunk is egg-shaped, the larger end being below. The pelvis as a region hardly exists, and the thorax is very small when compared with the large abdomen. The latter is very large, owing to the disproportionate development of the liver, presumably a great organ of nutrition during fœtal life. A striking peculiarity is the almost complete absence of shoulders, which with the arms are relatively insignificant outgrowths from the sharp end of the egg. It is evident that the small size of the thorax, its want of solidity, and the slight development of the pectoral and shoulder muscles indicate that its action in respiration must be very different from that in adult life.

The greatest breadth of the trunk is in the region of the lower ribs.

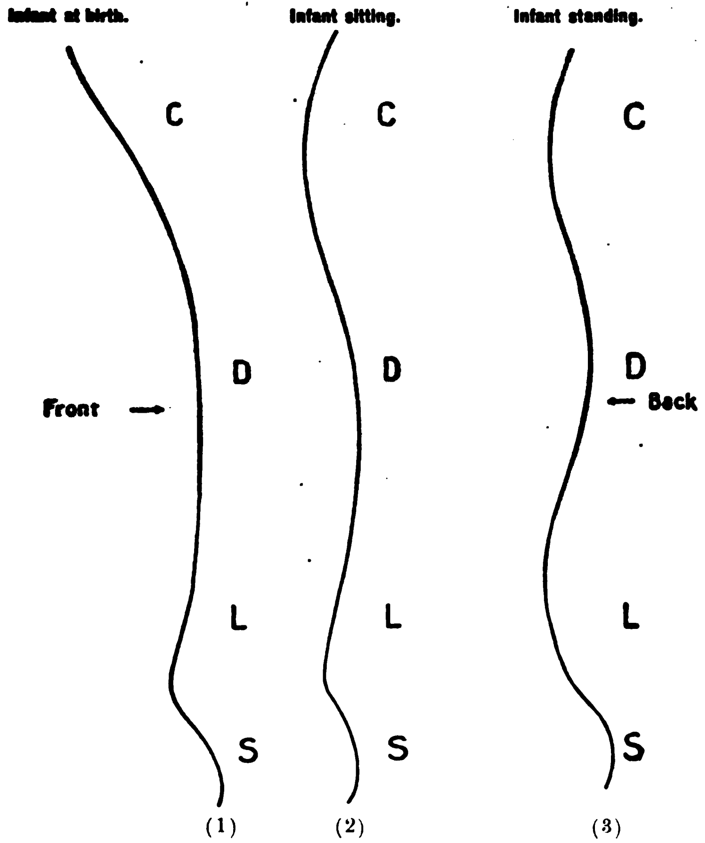
During intra-uterine life, and especially at the time of delivery, great flexibility and compressibility are requisite. Respiration has not yet occurred, and the assimilation of nutriment for the growth of the body and for preparing the rudiments of future organs has been the function most actively employed. When, therefore, we study the new-born infant, we must remember that we see it at an essentially transitional stage. Adaptations, the marked utility of which is past, still persist, and new functions are carried on with very imperfect apparatus.

SPINE.—At birth the spine consists of little bone and much cartilage and fibrous tissue. It can be twisted and bent at will in any direction. It appears relatively broader in proportion to its length than does the adult spine. The height of the vertebræ is relatively less, and appears even less than it is, from the fact that the broad, narrow, bony nucleus of the vertebral body, which catches the eye, does not represent the whole thickness of the body, as it is embedded in cartilage.

At this early stage of development the whole column is cartilaginous, with the exception of the nuclei of the bodies of the vertebræ and those of the laminæ on either side, forming a small portion of the body and the beginning of the arch. The time of the consolidation of the bodies is not accurately known.

Fig. 3.

SPINAL CURVES.



C represents cervical curve; D represents dorsal curve; L represents lumbar curve; S represents sacral curve.

In the young embryo, the proportion of the neck in the movable part of the spine is greater than that of the loins, a condition which is reversed in the adult, where the neck is less, being a little over one-fifth, and the loins a little less than one-third. In fact, the proportions of the spine change considerably from an early period of intra-uterine life to that of the perfected adult condition. At birth, however, the change has progressed sufficiently to make these two parts very nearly equal. The union of the laminæ to form the spine begins in the upper part of the spine sooner than in the lumbar region. Throughout the greater part they are nearly united, and in some places are quite joined, at birth.

Fig. 3 represents (1) the natural curve at birth, (2) the curve which comes especially in the *cervical* region when the infant has learned to sit up and the superincumbent head has to be supported, and (3) the additional *dorsal* and increased *lumbar* curves which appear when the child stands and walks, and which correspond to those of the adult condition.

At birth, when the child is lying in what may be called its normal position,—that is to say, on its side, with the head flexed and the thighs drawn up,—the whole spinal column presents one long concavity from the atlas to the coccyx, the front of which is subdivided into two curves by the slight projection of the promontory of the sacrum. Above this there is a tolerably regular concavity. The head can be thrown back so as to make a slight convexity in the neck, and by bringing the knees against the table (the infant being on its back) the lumbar region will spring forward; but the former of these positions is rather unnatural, and the latter impossible without assistance. We can then consider the part of the spine above the sacrum as essentially a fibrous and cartilaginous rod, with a number of separate disks of bone embedded in it at different places. The extent of the movements possible at birth, both in the dissected spine and in the whole body, is remarkable.

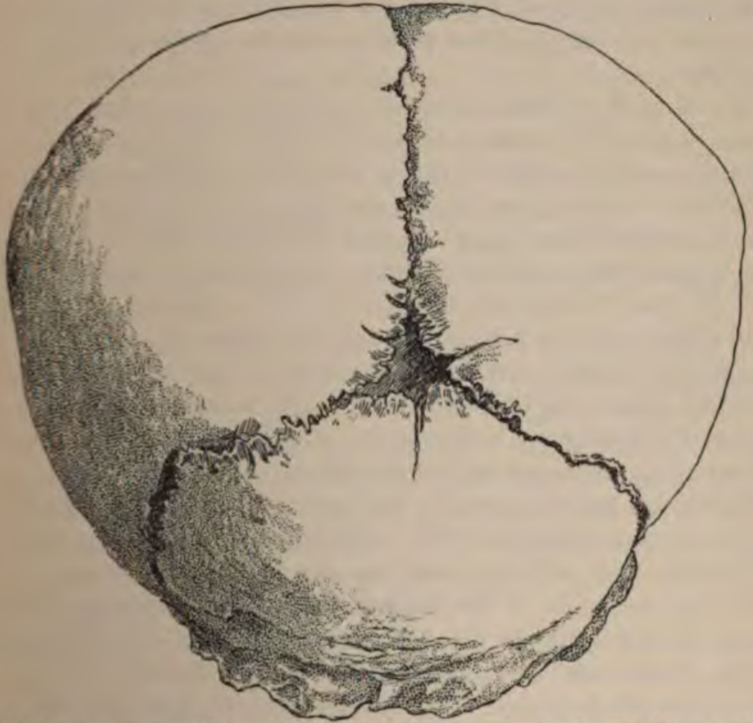
At all ages, from birth upward, the spine of the fourth lumbar vertebra is (as in the adult) on a level with the highest point of the crest of the ilium (Dwight). Under certain circumstances this might advantageously be used as a starting-point from which to count. At birth the spinal cord descends only the space of about one vertebra lower than in the adult. The third lumbar spine, which should mark its termination, cannot be easily recognized under three years, but the correspondence of the top of the ilium with the fourth vertebral spine allows its position to be estimated. It is desirable to know how far the cavity of the spinal dura mater descends inside the sacrum. Recent investigations by Wagner show that in children under a year old it ends usually near the top of the third sacral vertebra, which makes it a little lower than its usual termination in the adult. The point on the surface corresponding to this could be approximately estimated without any definite landmarks.

NECK.—It is customary to say that young babies have no necks; and yet when speaking of the spine it was stated that the cervical region of the vertebral column of the infant and young child is relatively longer than in the adult. From this point of view the shortness of the infant's neck must be seeming rather than real, but from a clinical stand-point it is real enough. The causes of the short neck are, first, the large head, which naturally falls forward, covering the upper portion, and next, the high position of the sternum encroaching on it from below. The large proportion of subcutaneous fat tends to make the neck appear still shorter.

The larynx is at first placed much higher than later (Symington). In the adult the lower border of the cricoid is about on a level with the top of the seventh vertebra. In the infant it usually seems to be near the lower border of the fourth vertebra.

HEAD.—Young infants frequently have at birth a startling shape to their heads produced by pressure. One side of the skull may be flattened, while the other bulges, or the natural diameters of the head may be altered, presenting a long, narrow head instead of a round, well-formed cranium. These shapes at times give an idiotic expression to the infant which causes much distress to the parents. In almost every case these abnormal appearances pass away in a few months, as the skull and brain grow, and do not, as a rule, indicate disease unless very extreme, so that it is well to state this fact to parents at once and thus to relieve their minds. As a rule, if the measurements of the head are taken over the middle of the forehead and around to the occipital protuberance, it will be found that at birth the circumference is about 33 cm. (13 inches).

FIG. 4.



Skull of infant at term, natural size. Posterior view, showing parietal and occipital bones and posterior fontanelle. Warren Museum, Harvard University.

FONTANELLES.—The opening between the frontal bones and the anterior borders of the parietal bones is called the *anterior fontanelle*, and, though somewhat depressed below the level of the bones at first, should soon be about on a level with them. Its size is variable, but is usually about 1 to 2 cm. (½ to ⅞ inch) long, and about 1 cm. (½ inch) wide. In the early days and even weeks of infancy the frontal suture is usually open in its upper part, as shown in Fig. 28, facing page 86.

The opening between the occipital bone and the posterior edges of the parietal bones is much smaller, is of less significance than the anterior opening, is often temporarily obliterated by the overlapping of the bones, and is called the *posterior fontanelle.*

FACE AND CRANIUM.—In infants the proportion of the face to the cranium is strikingly different from what is found in adults, where it is as one to two, while according to Froriep the face in the infant is to the skull as one to eight.

If the front view of the face and cranium of the infant and of the adult is contrasted by counting as face all below a line at the tops of the orbital arches, and as skull all that is seen above that line, considering it projected on a vertical plane as in a photograph, it will be found that in the infant the skull forms about one-half, and in the adult much less.

The height of the orbit bears pretty nearly the same proportion to the skull at all ages, except that it equals barely a third of the adult face, while it makes nearly a half of it at birth. While the top of the nasal opening retains pretty nearly the same relation to the orbit at all ages, its lower border is but very little below the lowest point of the orbit at birth, while it is much below it in the adult. In the latter, a line connecting the lowest points of the malar bones crosses the nasal cavity, or at least touches its lower border, while in the infant it runs almost half-way between the lower border and the edge of the alveolar process. The breadth of the skull in its greatest diameter in the infant equals, or even exceeds, the total height of the skull and the face, while in the adult it is but about three-quarters of it. Still more striking is the difference between the length and the breadth of the face at different stages. The breadth, measured between the most distant points of the zygomata, is to the height of the face in the adult about as nine to eight, while at birth it is perhaps as much as ten to four.

The side view is equally or even more characteristic. The auditory meatus is situated about midway between the front and the back in the infant, but in the adult it is decidedly behind the middle. The face appears to be but an insignificant part of the whole structure.

Jaws.—The lower jaw is almost on the same plane as the mastoid process of the temporal bone, and the upper border of the zygoma is about on a level with the floor of the nasal cavity, while in the adult it is at or near the level of the floor of the orbit. It is evident that a very important factor in the adult face is the development of the jaws and of the teeth, and that it is due to their rudimentary condition that the face is so small in infancy. The difference in the comparative development of the lower jaw at birth and at three years is well exemplified by the skulls shown in Fig. 12, page 49.

Gums.—The gums do not meet in the new-born (McClellan). They are composed of a dense fibrous tissue covered by vascular mucous membrane of very slight sensibility, and are protective to the growing teeth.

NASO-PHARYNX.—A median section of an infant's head shows very strikingly the want of height of the naso-pharynx and the great obliquity (approaching the horizontal) of the posterior edge of the vomer. The naso-pharynx is relatively very long from before backward. Strange as it may seem, the distance from the back of the hard palate to the soft parts of the back of the pharynx (excluding the tonsil) is about as great at birth as in the adult. A knowledge of the change in size and shape of the nasal cavities and *naso-pharynx* in the course of growth is very important. The nasal cavity consists of an upper olfactory region, occupying the ethmoidal portion of the cavity, and a lower respiratory region occupying the maxillary part. In the infant the nasal cavity is relatively long and shallow, and the respiratory portion is very narrow.

The height of the posterior nares at birth is 6 to 7 mm., and the breadth between the pterygoid processes at the hard palate is 9 mm. (Dwight.)

In the infant the naso-pharynx is simply a narrow passage running obliquely backward and downward from the constricted opening of the posterior nares. The soft palate of the child is placed more horizontally than in the adult. The posterior nares (not the inferior meatus alone, but the whole opening on either side) is just large enough to admit the end of a medium-sized male catheter; this leads into the passage just mentioned, and therefore a congestion of the nasal mucous membrane in infancy, with the addition of the mucous secretion, may effectually close the opening from the nose to the pharynx.

It is, perhaps, not sufficiently recognized clinically how important a function is performed by the nasal passages in early infancy,—far more important, indeed, than at any other age. In fact, the age of the infant is in inverse ratio to the dangers which may arise from obstruction of the nares.

These dangers, consequent on obstruction, congestion, and the resulting mechanical disturbance of neighboring parts, thus leading to actual disease of those parts, become in the new-born infant of most serious and even vital import. I have seen an infant die of simple acute nasal catarrh in the first two or three days of life. In this case the infant was, indeed, puny and ill cared for.

There is no doubt that, with due appreciation of the value of the nasal function and the danger of allowing it to be interfered with, we can, as a rule, even in extremely weak infants, prevent a fatal result.

Lymph-Vessels of the Pharynx.—An anatomical condition of great importance is that in comparison with the faucial tonsils, which are relatively poor in absorbents, an exceedingly rich plexus of absorbents exists in the posterior wall of the naso-pharynx.

Eustachian Tubes.—In the fœtus the nasal opening is below the level of the hard palate, which it reaches at birth. While in the adult the cartilaginous portion slants downward, nevertheless the opening of the tube is opposite a higher part of the nose than in the child. At birth the tube is horizontal or nearly so. It must be borne in mind that even if the opening of the tube be below the level of the hard palate, the soft palate none the less runs beneath it, shutting it off from the cavity of the mouth and the passage from it to the fauces.

In the infant and in the young child there is but a slight development of the end of the cartilage which makes in the adult so prominent a fold at the back of the pharyngeal opening of the tube, and by its prominence does much to determine the depth of the fossa of Rosenmüller, the recess behind it at the lateral posterior angles of the pharynx. At birth this prominence hardly exists. The opening of the tube is at first very small. That the catheterization of the tube at this age presents great difficulties of its own, apart from the intractability of the patient, is sufficiently obvious.

The tube in infancy, while of course shorter than in the adult, is stated to be not only relatively, but absolutely, wider at its narrowest point, which may explain the case with which catarrhal processes travel at that age to the middle ear.

Faucial Tonsils—Pharyngeal Tonsil—Lymphoid Tissue.—The faucial tonsils, the pharyngeal tonsil, the lymphoid masses under the mucous membrane of the posterior third of the tongue, the lymphoid tissue about the orifices of the Eustachian tubes, to say nothing of irregular aggregations of the same tissue in the neighborhood, form a lymphoid ring around the pharynx which is most important. It is to be noticed that the passage from the nose, as well as that from the mouth, is guarded by this apparatus. That its function is in part protective seems very probable, in spite of the fact that when hypertrophied it gives rise to serious trouble. Before birth this system is but slightly developed. Indeed, the follicles at the back of the tongue are not always to be found at that time.

A pocket in the pharyngeal tonsil is the famous *bursa pharyngea*. It is clinically important merely as a recess in which inflammation may linger and secretions be retained.

The supposition that this system is protective receives support from Killian's observation that the pharyngeal tonsil is much developed in mammals that live in the dust of houses. It is not impossible that this lymphatic ring forms a bulwark against septic invasion.

Goodale's investigations show that bacteria are found in the tonsillar crypts and that such organisms may be absorbed in the gland, but as soon as absorbed they meet with such conditions as would be likely to terminate their life. The small size of the naso-pharynx in the infant and the young child explains its obliteration by the enlargement of the pharyngeal tonsil.

MOUTH.—The mouth, as a whole, is pre-eminently an organ intended for the reception of a liquid food, its mechanism being that of suction. It is a natural and necessary passage-way to the organs of digestion, but is not at first intended to aid the digestion by a salivary secretion.

Tongue.—The tongue at birth is slightly coated and comparatively dry. It is greatly wanting in vertical thickness, and on section is shown to be long and narrow. The soft palate, therefore, rests on the tongue, and, the mouth being closed, runs in the main backward, descending very much less than in the adult. The uvula is rudimentary (Merkel). Owing to the depth of the pharynx (from before backward), the soft palate is unable to shut off the passage to the naso-pharynx as completely in early infancy as subsequently.

Teeth.—At birth there are twenty embryo teeth, ten in each jaw, enveloped in their respective tooth-sacs, protected above by the submucous tissue and mucous membrane, on either side by alveolar bone-substance, and below by the groove in the maxillary bone from which the alveoli have developed.

HARD PALATE.—At birth and in the early months of life the line of the hard palate, continued backward, strikes near the top of the basi-occipital, that is, near its junction with the sphenoid, or perhaps even strikes the latter. Accordingly, at this age, if the finger be introduced directly backward through the mouth, pushing the soft palate upward, it will strike the occipital bone, and being carried a little downward, will pass over the arch of the atlas, the base of the odontoid, and the body proper of the axis. Going still lower, the top of the third cervical vertebra might be felt, but the larynx would hardly permit the finger to go lower, and the parts are so small that it is questionable if much could be recognized below the axis.

BRAIN.—The brain of the new-born infant is proportionately very much larger than in the adult, bearing a relation of about 15 to 1. (Vierordt.)

EYE.—The eye is anatomically perfectly developed in the new-born. (McClellan.)

EAR.—The development of the ear is in its several parts very unequal (McClellan). The structures of the internal ear, the tympanic cavity, and the auditory ossicles are fully formed at birth, while the external auditory meatus, the Eustachian tube, and the mastoid portion of the temporal bone undergo many modifications before their full development at puberty. At birth the meatus passes inward and inclines downward, and the membrana tympani is almost horizontal, conditions to be remembered as necessitating a little different management of the ear speculum from what is taught in the examination of the adult ear.

The mastoid antrum exists at birth, but the cells do not develop until later.

Petro-Squamosal Suture.—An important anatomical condition existing at birth is, that the *petro-squamosal suture* is open, allowing a close connection between the blood-vessels of the brain and the middle ear, with its resulting clinical significance.

THORAX.—The thorax of the infant forms the upper and smaller end of the egg-shaped body which has been already described, while the small shoulders make the chest very different from that of the adult. Besides this, the whole shape of the thorax is very peculiar. The proportion of the dorsal region of the spinal column is pretty nearly the same throughout life, but the thorax itself varies greatly. At birth the thorax is very insignificant. In front the breast-bone is relatively much smaller than that of the adult male, but not very different from some very small breast-bones which are occasionally met with in women. The lower part is, however, but slightly developed. The borders of the ribs diverge relatively rapidly. This is perhaps due to the great breadth of the abdomen.

Top of the Sternum.—The sides of the thorax are not relatively so long as in the adult, which is probably partly due to the lesser development of the lower ribs and partly to the very important characteristic of the infant's thorax,—namely, that the top of the sternum is placed higher than in the adult. The top of the sternum in the latter is about on a level with the disk between the second and third dorsal vertebræ. The top of the sternum, according to Symington, is opposite about the middle of the first dorsal vertebra in the new-born infant, and a frozen section by Rüdinger shows it to be rather below the middle of the first.

Diameters.—Another most important peculiarity of the infantile and child's thorax is its want of breadth. In the adult throughout the thorax, from about the level of the second costal cartilage, or even a little higher, to the top of the diaphragm, the antero-posterior diameter of the interior of the thorax is to the transverse as one to two and a half or one to three, there being, of course, a certain amount of variation. At birth, on the other hand, it is as two to three.

It is well known that in the infant the ribs are more nearly horizontal than in adult life. A striking feature of the young infant's chest is that the ribs form the sides of the chest, and the sternum and cartilages the front.

Ossification.—At birth the sternum is practically a strip of cartilage in which a varying number of bone-centres have been deposited. There is one for the manubrium and usually one or two for the second and third pieces, those for the latter being very frequently double. These, however, are mere thickenings of the cartilaginous strip, which is flexible and pliable in all directions. (Figs. 28 and 29, facing page 86, show these divisions of the sternum.)

Respiration.—An important feature in the mechanism of thoracic respiration is the rigidity of the thorax. In the infant at birth this rigidity is almost wholly absent, as it is found only in the ribs.

The sternum is at this age practically a perfectly flexible strip of cartilage, for the small points of ossification in it only modify the softness of certain separate parts. The dorsal region of the spine is not fixed as a concavity, but can be bent freely backward. The motions of the ribs are practically the same as in the adult, but the effect of these motions is different. In the first place, as has been shown, the ribs are more nearly horizontal, and the thorax, even after death, is in what is called the inspiratory condition. The nearly horizontal first rib can hardly rise any higher unless the whole spine is bent backward. The ribs, being straighter than in the adult, do not when raised increase the breadth of the chest to the same degree. The nature of the infantile respiratory movements is far from easy to analyze. Sometimes it seems abdominal and sometimes thoracic. The fact is, that at first it is of a very indefinite type. The thorax seems to expand as it can. It is common to see its lower part drawn inward by the contraction of the diaphragm. At birth no especial part of the respiratory apparatus has attained a sufficient development to insure its continuous equable action, and, as would be expected, irregular respiratory movements and no decided type of respiration are found.

A sufficient number of observations, however, have not yet been made to warrant our stating any especial age at which the type of respiration in the two sexes separates and the female infant assumes the thoracic type of respiration. But if the breathing of the infant is essentially irregular in type, it is admirably adapted to the wants of its age. The elastic thorax can give way under pressure and expand in almost any direction. The flexible sternum submits to liberties which no adult breast-bone would endure. One-half of the chest may be compressed and yet the other go on acting independently.

The facts that the top of the sternum is higher, reckoning from the spine, that the ribs are more nearly horizontal, and that probably the lower part of the sternum is relatively less developed than in the adult,—necessarily imply certain peculiarities in the relations of the internal parts.

DIAPHRAGM.—The diaphragm rises highest on the right over the summit of the liver, is a little lower on the left, and lower still at its tendinous centre in the median line. It is generally stated that the diaphragm is higher in the child than in the adult. Dwight's observations, partly original, partly on the frozen sections of other writers, give the following results. In the infant the diaphragm appears to be opposite the disk between the eighth and ninth dorsal vertebræ. In the infant it appears as if there were a lower insertion of the diaphragm to the sternum and the seventh costal cartilages than in the adult. Usually the line runs from one costal arch to the other, somewhat above the apex of the ensiform cartilage, leaving, therefore, a space on either side of the latter, where the interior of the thorax is against the abdominal walls. Owing, perhaps, to the greater flexibility of the body and to the less firm attachment of the internal parts one to another, it certainly seems that at least after death the thoracic cavity is more accessible at the sides of the ensiform than it is in the adult.

We may conclude that, while there is some variation, on the whole, the central point of the diaphragm is in the infant higher in relation to the spine than later in life, and that it gradually becomes lower. How high the diaphragm rises laterally is hard to say, for it is a point very difficult to observe. According to Kölliker, in the fœtus at term, on the right it reaches the level of the anterior end of the fourth cartilage, and on the left that of the fourth intercostal space. Henke adds to this that certainly after respiration has begun it will never be so high again.

Thymus Gland.—The thymus gland exists at birth, and lies above and to some extent before the heart.

Heart.—The most striking peculiarity of the infant's heart is that it is less covered by the lungs than in adult life. Together with the thymus gland it forms a solid mass from the posterior mediastinum to the sternum, pushing the lungs far apart. It is to be noticed, however, that the pleural cavities extend as far forward as in the adult. The relations of the heart to the chest walls are curious in the infant, for these anterior walls are, as already stated, high in relation to the spine, yet the heart itself is high in relation to the walls. At least the upper half of it is so. With regard to the apex and the lower borders the relations are less certain. We usually find the impulse of the heart rather higher and nearer to the mammary line in the infant than in the adult. The weight of the heart at birth is 20.6 grammes (about ⅔ ounce), according to Boyd, and its proportion to the rest of the body is largest at about the time of birth.

Common Carotid Artery.—The common carotid artery has in the new-born half the length of the descending aorta, but this proportion is much lessened at a more advanced age, when the vertebral column increases in length.

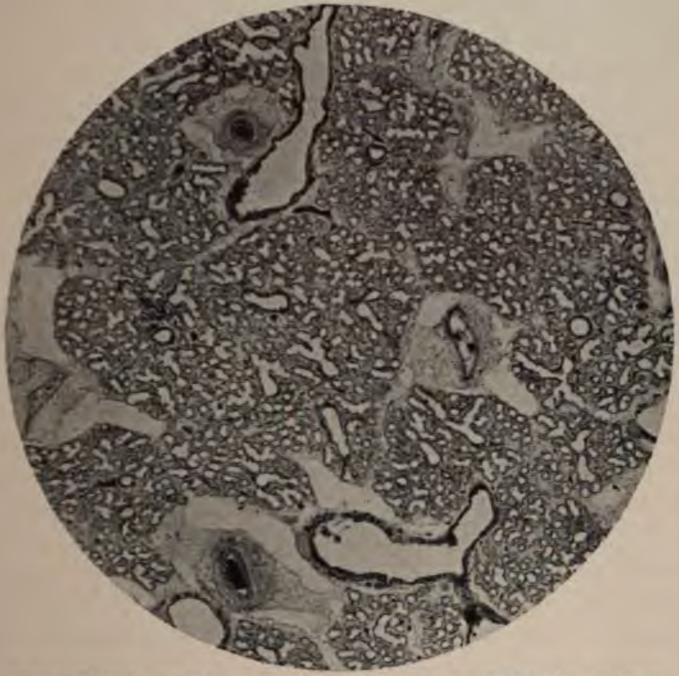
Veins.—According to Jacobi, there are one hundred valves in the veins of the lower extremities of the new-born.

Pulmonary Artery.—The pulmonary artery also, as stated by Jacobi, is from two to four centimetres (three-fourths to one and five-eighths inches) larger than the descending aorta.

Lung.—Anatomically on the right side the eleventh rib behind marks the lower border of the lung, while it descends as low as the twelfth rib on the left side. In front the lung extends to about the fourth or fifth rib on the right side and the sixth rib on the left side. The lung at birth is characterized by its embryonic type. The infant's lung represents an intermediate condition of growth, which illustrates the gradual change from the fœtal to the adult condition. (Figs. 5 and 6, Northrup.)

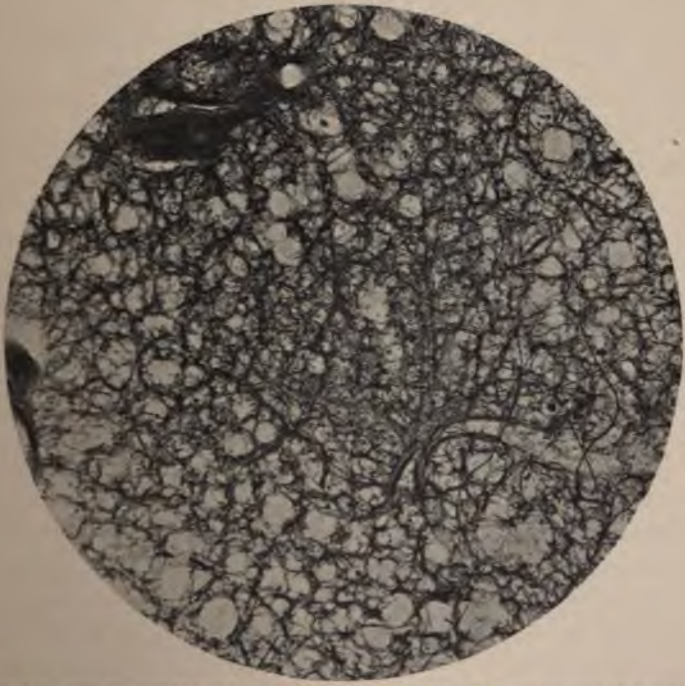
According to Northrup, if we examine the lung of a five months' fœtus it is found that the bronchi constitute the entire respiratory tract thus far developed. At the terminal end of the bronchi there are bud-

Fig. 5.



Section of fœtal lung at 5 months, showing development of bronchi; no alveoli.

Fig. 6.



Section of infant's lung at 10 months, showing increased proportionate amount of parenchyma in comparison with the fœtal condition; distended alveoli.

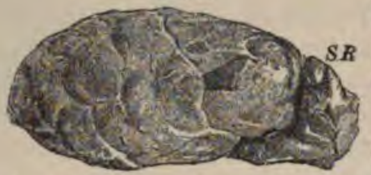
like dilatations, which are the rudimentary air-spaces. Between these dilatations, and separating them from each other, is loose, delicate connective tissue, which makes up the remaining bulk of the lung, so that what subsequently becomes the alveoli is about equal in extent to the previous bronchial development. This rudimentary air-space is destined to enlarge, subdivide, and finally, in early adult life, to occupy all the available room among the bronchial branches. The loose connective tissue becomes finally thin, dense bands constituting the stroma. This serves to distribute the vascular net-work, and upon this are laid the close-fitting epithelial linings of the air-spaces. In fœtal life the mucous membrane lining the bronchial tubes is loosely attached to the muscular walls, and is commonly seen lying in wavy folds within the contractile ring, where the same delicate connective tissue loosely holds the growing tissues together. As has been said, the aërating portions of the lungs develop as bud-like dilatations at the tips of the smallest bronchi. These dilatations in the course of their development extend into the stroma. The epithelium, changing from the columnar type characteristic of the smaller bronchi, covers the newly made walls with flat respiratory epithelium. At birth the loose connective-tissue stroma of the fœtal lung of five months has been condensed into rather thick alveolar walls. Another feature of the child's lungs as contrasted with those of adults is the behavior of the blood-vessels. Being loosely restrained in the walls, they easily become distended and tortuous and encroach upon the cavity of the alveoli. With small alveoli, thick walls, and abundant distribution of vessels, it is easy to understand how, in hypostasis, distention of the vessels may be an important factor in displacing the air in feeble subjects with weakened respiratory vigor and partially obstructed bronchi. Finally, the lung of the infant differs from that of the adult mainly in the following respects. Proportionately the extent of the bronchial tubes is greater than that of the air-spaces. The connective-tissue stroma is likewise in greater abundance and tends to cellular proliferation. The submucous connective-tissue of the bronchi is loose and more abundantly supplied with nuclei, and its vessels are held more loosely. The cells lining the air-spaces form a continuous layer. The alveoli are small, their epithelium proliferates abundantly, and the absorbents accomplish their work slowly, the blood-vessels playing a more important rôle. These facts are to be borne in mind in connection with the bronchial lesion which forms so important a part of bronchopneumonia.

ABDOMEN.—The essential differences between the abdomen of the infant and that of the adult are the great size of the *liver* in the former, and also the relatively large size of the *kidneys* and the *suprarenal capsules*.

The liver, especially on the right side of the abdomen, encroaches on the space which is later occupied by other organs. Its relative weight to that of the whole body at birth is about 1 to 18. (McClellan.)

On the left side of the abdomen these conditions are not of much importance, but on the right, occurring as they do in connection with the great size of the liver, the large kidney occupies a lower position, and thus still further curtails the free space in the right flank. Viewed from the stand-point of the adult condition the relations are, as has been pointed out by Henke, much more peculiar on the right than on the left. The kidney as a whole is lobulated, as seen in Fig. 7.

Fig. 7.



Lobulated kidney, natural size. Infant three days old. *S R* marks the suprarenal capsule. Warren Museum, Harvard University.

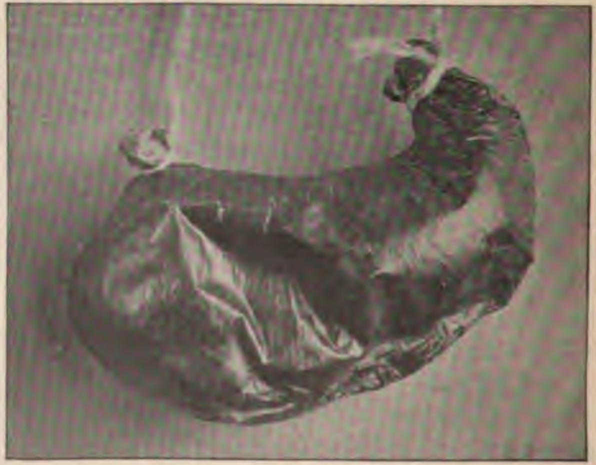
Uric Acid Infarction.—At birth a prenatal condition, represented by an orange or a light red colored deposit near the pyramids in the straight tubules of the kidney, exists normally. This condition is called the *uric acid infarction*, and the deposit consists of urate of ammonium, amorphous urates mixed with uric acid crystals, and some epithelial cells. (Plate III., 3 and 5, facing page 84.)

The suprarenal capsules at birth quite cover and surmount the kidneys, as seen in Fig. 7.

STOMACH.—The stomach at birth is remarkably small, and more tubular than in the adult, the fundus being but slightly developed. It is consequently even more vertical than in the adult, for it is the enlargement of the greater *cul-de-sac* that makes the obliquity of the axis pronounced.

Fig. 8, representing a stomach taken from an infant three hours old, shows very well the organ at birth. Its capacity was 25 c.c. ($\frac{5}{6}$ ounce). The weight of the infant was 2500 grammes ($5\frac{1}{2}$ pounds). Although the weight was below that of the average infant at birth, the stomach was of about the average size, as was shown by its gastric capacity.

Fig. 8.



Stomach, natural size. Infant three hours old. Warren Museum, Harvard University.

DUODENUM. — The duodenum, in the adult, has of late usually been described as ring-shaped, but it generally presents pretty well marked angles, which divide it into a horizontal part running backward, a descending one along the right side of the spine, a transverse one crossing usually the third lumbar vertebra, and, finally, an ascending part along the left of the spinal colunm, which brings the end to about the same level as the beginning. Sometimes the last two parts are represented by a single one running obliquely upward to the left, in which case the duodenum is called V-shaped. The first horizontal portion is often somewhat dilated, and its walls are smooth, the folds beginning usually with the descending portion. The walls of the duodenum just beyond the pylorus are lined by a continuous layer of Brunner's glands, which extends through the first part, becoming more or less broken up towards the end. In the infant the shape of the duodenum, as shown by plaster casts (specimens in Warren Museum), is more nearly that of a ring, the two lower angles being rounded off. A constriction is often (perhaps usually) seen at the junction of the first and second parts, but Dwight's casts of the infant's duodenum do not show the folds, which are very striking in the casts taken from adults. That is to say, those of the infant show a few deep cuts into the cast instead of a great many near together. I have seen the folds, however, very richly developed in an infant of three weeks. In one case, that of a female six weeks old, Dwight found the duodenum of the V-shaped pattern, and, what is more remarkable, after it had passed the gall-bladder it was surrounded by peritoneum so as to swing freely as a loop suspended from the posterior abdominal wall. As to Brunner's glands, a few observations on young children have suggested that they were rather less developed relatively than in the adult, but this may not always be the case. The duodenum has been compared to a trap, its ends being always higher than its middle, which is thus fitted to retain the fluid poured into it from the liver, the pancreas, and its own glands, besides that which it receives from the stomach.

The number and size of the folds and the shape of the duodenum in the adult would tend to delay the passage of its contents through it, and thus it also prevents the passage of gases from the small intestine upward into the stomach. If it be true that in the infant the system of folds is less developed, its passage would be relatively easy, which with a fluid diet seems desirable.

INTESTINES.—From what we know of the development of the intestinal tract, which was at first merely a loop loosely attached to the posterior abdominal wall, it is natural to expect that in the infant and young child it should be less fixed than in adult life; and this is, in fact, the case. The difference is most striking in the large intestine, and is shown particularly in the cæcum, ascending colon, and sigmoid flexure. That this condition gives rise to dangers is evident, and I should say that there is a strong probability that the cases of infantile intussusception which occur with unusual frequency during the middle of the first year may arise from this anatomical peculiarity, and this makes a thorough knowledge of the anatomy of the intestine important. In the fœtus at full term the length of the intestine, and especially of the colon, is singularly constant.

Small Intestine.—The average measurement of the small intestine in infancy is 287 cm. (9 feet 5 inches). The variation may amount to 61 cm. (about 2 feet).

Large Intestine.—The large intestine at birth, according to Treves, measures 56 cm. (about 1 foot 10 inches). So regular are these measurements that the greatest variation that I have met with in the colon was as little as 12.7 cm. (about 5 inches).

Sigmoid Flexure.—But little of the sigmoid flexure is found in the pelvis at birth.

PELVIS.—The small size of the infant's pelvis is to be noted also as the cause which, to a greater or less extent, forces the pelvic organs of later life into the abdomen during infancy.

BLADDER.—In the infant the bladder is practically wholly an abdominal organ.

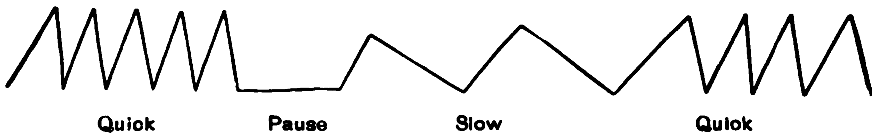
UTERUS.—At birth, part of the uterus is above the brim of the pelvis.

TEMPERATURE.—The temperature at birth is slightly higher than a few days later. It is about 37.2° C. (99° F.).

PULSE.—The pulse varies from 120 to 140 to the minute at birth, and it is at times irregular, especially during the first few hours.

RESPIRATION.—The respiration is about 45 to the minute, but it is of a very irregular type, and the rhythm changes continually. The breathing is superficial, sometimes quick, and again dying away so as to be almost imperceptible. This condition, if occurring in an older child, would be a symptom of grave disease, but may be said to be normal at birth. The rate may be much quicker than 45, and I have frequently observed it as high as 60 or 70.

Fig. 9.



Respiration at birth for one-fourth minute. Awake, but quiet.

HEIGHT.—The new-born infant's average height is in the male about 49.5 cm. (19¾ inches); in the female 48.5 cm. (19¼ inches).

WEIGHT.—The weight of the male infant is usually rather greater than that of the female. The average weight in a large number of cases showed that of the male to be 3250 grammes (7⅐ pounds), while that of the female is 3150 grammes (7 pounds). Parker, in a careful examination of 170 infants at birth, of whom 89 were males and 81 females, found that the average weight of the males was 3520 grammes (7¾ pounds), while that of the females was 3290 grammes (7¼ pounds). There is, then, a certain amount of latitude to be accepted in this question of weights.

The weight, however, has so close a connection with the vitality of the infant, that although we often see infants of light weight vigorous and thriving, and those of considerable weight failing to gain, yet as a general index of vitality the weight is a valuable starting-point and guide for our treatment. Rules and averages of this kind are not to be depended upon absolutely, but simply represent conditions which with other important factors aid in solving the problem of vitality.

VITALITY.—In the early hours and days of existence it is the disturbance of the equilibrium of the infant's vitality which is especially to be feared and combated rather than the specific morbid processes of later childhood. We should therefore in each infant carefully determine the degree of inanition which we are called upon to deal with at this period of life, and I have personally found it useful, as shown in the following table, to divide the weak and strong infants into groups according to their weights, allowing a somewhat lighter weight for girls than for boys.

TABLE I.

Relation of Weight to Vitality.

Groups.	Weight.	Vitality.
1	2000 grammes (about 4½ pounds)	Very low.
2	2500 " " 5½ "	Low.
3	3000 " " 6½ "	Fair.
4	3500 " " 7½ "	Normal.
5	4000 " " 8 "	High.
6	4500 " " 9 "	Very high.

HANDS.—At birth it is quite remarkable to find with what manifest strength the infant can grasp your finger. The nails are well formed.

FEET.—A very important part of the infant's anatomy is the foot. The later researches of Dane have shown that the foot of the new-born infant is not normally flat. On the contrary, the bones are so arranged as to form an arch that is even higher in proportion to its length than that found in adult feet. The flat appearance is caused by the presence in the sole of a very large amount of fat tissue and the greater proportionate size of the adductor pollicis muscle.

BONE MARROW.—At birth, and in the early months of life, the marrow of the bones is red, as shown in Plate II., facing page 80.

The red color caused by the numerous injected blood-vessels is more intense at the central portion of the section of this bone than at the periphery or towards the ends.

FUNCTIONS.—Voice.—The normal infant at birth should present a developed voice, and should cry vigorously, thus assisting the lungs to expand and the new circulatory mechanism to be well started.

Sight.—Although the eye is anatomically developed and is sensitive to light, and although the visual perception is also possibly developed, yet there is still a lack of power to interpret the images perceived.

Hearing.—The auditory sensations appear to be rather dull during the first few days of life. This is possibly due to the absence of air from the tympanum and a tumid condition of the tympanic mucous membrane.

Touch.—The sense of touch is well developed.

Taste.—The sense of taste is well developed.

Smell.—The sense of smell is probably well developed; but this is still a matter of dispute.

Sebaceous Glands.—The function of the sebaceous glands is fully developed at birth.

Lachrymal Glands.—The secretion of the lachrymal glands is not developed at birth. The new-born infant does not shed tears, a fact of some clinical consequence in connection with the prognosis as to the convalescence of disease in the early days of life.

Sweat Glands.—The function of the sweat-glands is not developed at birth as a rule, but according to my observations perspiration in certain individuals certainly occurs at a much earlier period than is usually supposed. I have seen an infant, premature at the seventh month, perspire freely one week after it was born, and in a number of individuals this function must exist in the early days of life.

Salivary Glands.—The salivary secretion is not fully established at birth, and consequently the mucous membrane of the mouth is comparatively dry. The peculiar whitish color of the young infant's tongue is caused by the epithelium not being washed away by the saliva to the extent that it is after the later development of the function of the salivary glands. The amylolytic function of the saliva is very slightly present at birth. The amylolytic action is indeed so insignificant that it merely shows us that the function of the salivary glands in the early months of existence is only partially developed and certainly should not be called into use.

Pancreas.—The amylolytic action of the pancreatic secretion at birth is probably not all developed. The fat digestion is fairly developed at birth. The proteid digestion is fairly developed, but not fully.

Bile.—According to Foster, "the excretory functions of the liver are developed early, and at about the third month of intra-uterine life bile-pigment and bile-salts find their way into the intestine. A quantity of bile secreted during intra-uterine life accumulates in the intestine, especially in the rectum, and forms, together with the slighter secretion of the rest of the canal and some desquamated epithelium, the meconium. The distinct formation of bile is an indication that the products of fœtal metabolism are no longer wholly carried off by the maternal circulation, and that to the excretory function of the liver are now added those of the skin and kidney."

BLOOD.—It is impossible by the methods at present known to determine exactly the total amount of blood in either infant or adult, but, while the adult's blood is approximately about one-thirteenth of the entire weight of the body, the infant's is represented by only one-fifteenth. A full description of the normal blood in infancy and childhood will be found on page 874.

LYMPHATIC SYSTEM.—The lymphatic system is very active at birth.

URINE.—The amount of urine secreted during the first two days of life is very small, and its specific gravity is about 1010. The kidney shows the condition of the uric acid infarction, and it is not infrequent to find the napkins stained with a uric acid deposit, such as is represented in Plate III., 3 and 5, facing page 84.

INTESTINAL DISCHARGES.—Unless a discharge of the contents of the intestine has taken place during the delivery, as is so often seen in breech presentations, it occurs immediately or very soon after birth.

MECONIUM.—The discharge which first comes from the intestine is called the *meconium*. It is inodorous, viscid, slightly acid, and of a brownish-black color, such as is represented in Plate III., facing page 84. The meconium contains mucus, epithelium from the intestinal mucous membrane, epidermal cells, hairs, and fat-drops from the vernix caseosa which have been swallowed with the amniotic fluid from time to time. It also, according to Vierordt, contains the constituents of the bile, and its total amount is from sixty to ninety grammes (two to three ounces), of which the solid part forms about twenty per cent. The intestinal contents at birth are sterile.

IV. NORMAL DEVELOPMENT.

In order to differentiate normal from abnormal conditions in the growing infant and child, the different stages of development corresponding to the various ages should be studied.

CORD.—By a process of disintegration the cord at about the seventh or eighth day separates from the living tissues around the umbilicus. A certain amount of bleeding may take place at the point of separation, but this is usually very slight; it may, however, be the beginning of one of the most serious forms of disease in the new-born, *umbilical hemorrhage*. The umbilical depression is well marked, even when the infant cries, and this normal anatomical condition following the separation of the cord can thus be distinguished from the umbilical prominence representing cases of umbilical hernia.

SPINE.—The time of consolidation of the bodies of the vertebræ is not accurately known, but it may be roughly stated to begin in the third year, and, probably, to end in the seventh. A large number of observations must still be made before the various stages of ossification can be determined. The statements regarding this point are copied from one book to another, and are often quite imaginary.

The union of these chief centres to form the bodies of the vertebræ begins in the lumbar region, and is first completed there. This union, however, had not taken place in the dorsal and cervical region of the child said to be three years old, used for "The Frozen Sections of a Child" (Dwight). On the other hand, in a girl of five or six years, figured by Symington, the process was found to be hardly finished in the lumbar region, and higher up it seemed about the same as in the younger child. The process of union of the laminæ is probably completed in the first few months of life.

LENGTH.—The following table shows the results of the measurements of the spines of children by various authorities. There is a remarkable uniformity of observations by different men in spite of the errors incident to the personal equation of the investigators and the individual variation which doubtless exists. The relative length of the dorsal (more properly the thoracic) region is shown to be somewhat greater than that of the adult; still it appears that after the age of five or six the proportions are not far from those of after-life.

TABLE 2.

Length of Spine to Sacrum.

ABSOLUTE LENGTH. IN MILLIMETRES.						RELATIVE LENGTH. Total = 100.		
Age.	Observer.	Cervical.	Dorsal.	Lumbar.	Total.	Cervical.	Dorsal.	Lumbar.
3 months....	Rasenel....	50	100	58	208	24	48.1	27.9
6 months....	Aeby......	52.5	103	60	215.5	24.3	47.5	27.8
6 months....	Aeby......	53.5	107	61	221.5	24.1	48.6	27.5
10 months....	Dwight....	61	125	77	263	23.2	47.5	29.2
2 years, boy..	Rasenel....	70	140	90	300	23.3	46.7	30
2 years, boy..	Aeby......	79.5	153.5	98	331	24	46.4	29.6
3 years, girl..	Dwight....	78	162	101	341	22.9	47.5	29.6
4 years, girl..	Aeby......	79.9	162	103.3	345.2	23.1	46.9	29.9
5 years, boy..	Symington .	80	170	104	354	22.5	48	29.4
5 years, boy..	Rasenel....	80	180	135	395	20.3	45.6	34.2
6 years, boy..	Symington .	80	175	106	361	22.2	48.5	29.3
9 years, girl..	Rasenel....	85	195	150	430	19.8	45.4	34.9
11 years, boy..	Aeby......	91	218.7	153.5	463.2	19.7	47.2	33.1
13 years, girl..	Symington .	95	220	136	451	21.5	48.7	29.1
16 years, girl..	Aeby......	100	221.9	151	472.8	21.1	46.9	31.9
16 years, girl...	Aeby.......	107.5	229.5	152.5	489.5	21.9	46.9	31.1
17 years, girl..	Dwight....	113	250	161	524	21.5	47.7	30.7

The figures to the left of the double line in the table express the absolute length of the different portions of the spine, in millimetres.

Those to the right are the same figures reduced to terms of 100, within a fraction.

FLEXIBILITY.—The spine is very flexible at birth. This flexibility becomes less as the infant grows older.

In the cadaver of a female child of ten months it was found that extension was no longer so free as in the earlier months, and it required a strong pull to make the head touch the nates. The dorsal region, however, could still be made concave behind. Flexion was free, especially in the lower part of the lumbar region, where the pelvis and legs could be swung forward. On rotation the head could be turned through an arc of 90° without using the joint between the atlas and the axis. In a male child of the same age, extension of the spine was found to be still more restricted.

CURVES.—At birth there are no natural curves except the sacral in the infant's spine. An important factor in the production of the curves in the cervical and dorsal regions is probably the pull of the muscles. The dorsal curve seems to be a permanent condition of a part of the general curve of the body. As soon as the muscles of the back of the neck contract so as to raise the head from the chest, the front of the neck will be convex, and finally this becomes the habitual position. As Symington has pointed out, however, this cervical curve is never, properly speaking, consolidated, for it can always be obliterated by a change of the position of the head. The production of the lumbar curve is more complicated. If an infant be laid on its back on a table, the knees are raised and fall apart; if they are brought together and forcibly pressed down, the lumbar region will spring up from the table and the beginning of a lumbar curve will appear. It is supposed that this is caused by the shortness of the ilio-femoral ligaments, which, when the thighs are brought down, flex the pelvis, throwing the promontory of the sacrum forward. As the child begins to stand, the body is inclined forward, and when this is straightened by the muscles of the back the same thing occurs, for of course it is unimportant whether the legs are extended on the trunk or the trunk on the legs.

This curve, therefore, is first observed when the child is one or two years old, but it is not until some time later that it is habitually present. It can be obliterated up to adult life. The tonicity of the muscles has a great deal to do with retaining the curves of the spine and with limiting its movements. The importance of the muscles in distortions is very great. The spine of the child is flexible in many ways, and the unruly pull of a muscle may easily produce a lasting effect. Not only should the muscles have strength enough to maintain the figure without conscious effort, but their action should be symmetrical on both sides, and should also have a proper relative force before and behind. The importance of light gymnastic exercises is now generally understood.

Fig. 3, page 23, represents the curves of the infant's spine at birth and also at different ages up to the period of standing.

SURFACE ANATOMY.—The surface anatomy of the spine is of much importance in the child. In the infant, except perhaps in the neck, the back is rounded; later it is more flattened, and the line of the spinous processes is rather prominent. When we examine the dissected spine from behind we find it very different from that of the adult. In the infant the laminæ look more directly backward, and their presence in the median line is marked by knobs and ridges very different from the spine of the adult. Up to a year, or perhaps eighteen months, the proportions are not very different, but the spine at three years shows that a great change has occurred, for the spinal processes now stand out in a prominent row, and present very nearly adult proportions. The greatest difference is in the dorsal spines, which are relatively broader at their points and less gracefully drawn out than in the adult. The bodies of the vertebræ still remain less deep, and therefore the relative positions of the spines and bodies show less difference than might be expected. At six or seven years the spine has made still further progress towards the adult proportions. By the end of the second year the back of the living child is not only flatter and broader (the results of continuous changes), but there is the appearance of the median furrow, and at five or six the differences in this respect from the adult are not marked. It is barely possible to count the spines in the infant and young child, and at three and four years it is not very easy, though less difficult than in the adult.

The first dorsal spine, and not the seventh cervical, is the most prominent in that region. The atlas has no spine at all; the spinous process of the axis is thick and prominent, perhaps relatively less marked in the child than in the adult; the third and fourth spines are very small; the fifth is not much larger; but the sixth projects more, and the seventh is said to be usually the first prominent one. The relative size of the lower cervical spines varies considerably. The sixth may be the first to assume prominence, and the seventh cervical and first dorsal may exceed it but little. It is easier to examine a child of three years and upward than an adult, on account of the greater softness of the tissues, which allows us to feel more deeply in through the furrow of the neck, and, having recognized the axis by alternately flexing and extending the head, to count the cervical vertebræ in order. If it should be in any case absolutely impossible to feel the third and the fourth, it is better to allow a certain space for them and to call the next one the fifth than to assume arbitrarily that a certain one is the seventh. Confirmatory evidence may be gained from the height of the sternum.

NECK.—(See also p. 24).

CRICOID CARTILAGE.—Symington states that in two children respectively five and six years old the lower border of the cricoid cartilage was found to be at the lower border of the fifth or at the top of the sixth vertebra. The position of the head, in his observations at intermediate ages, in these measurements varied a good deal. In a girl of thirteen he found that it had reached the adult position; that is, about on a level with the top of the seventh vertebra.

EPIGLOTTIS.—Symington found also that the top of the epiglottis descends during growth from about the level of the lower border of the atlas to the middle of the third cervical vertebra, or even lower.

LARYNX.—This high position of the larynx would imply a greater part of the trachea relatively above the sternum, but this is neutralized by the

high position of the latter. The amount of fat in the neck makes the trachea less accessible. The greater distance of the trachea from the surface, as it descends, and the greater danger of meeting the large arteries and veins above the sternum in the child, are points of anatomy so well known in connection with tracheotomy that it seems hardly worth while to dwell upon them.

DISTANCE FROM CRICOID TO STERNUM.—A condensation of Tillaux's statements of the distance from the cricoid to the sternum is given in Table 3.

TABLE 3.

Relation of Cricoid to Sternum.

Years.	Distance from Cricoid to Sternum.
2½	3.5 centimetres.
3	4 "
3½	4 "
4	3.8 "
4½	4 "
5	4.5 "
6	4.9 "
6½	5.5 "
7	5.1 "
7½	4.5 "
8	5 "
8½	5.25 "
9	5.25 "
9½	6.5 "
10	6.5 "
10½	6.5 "

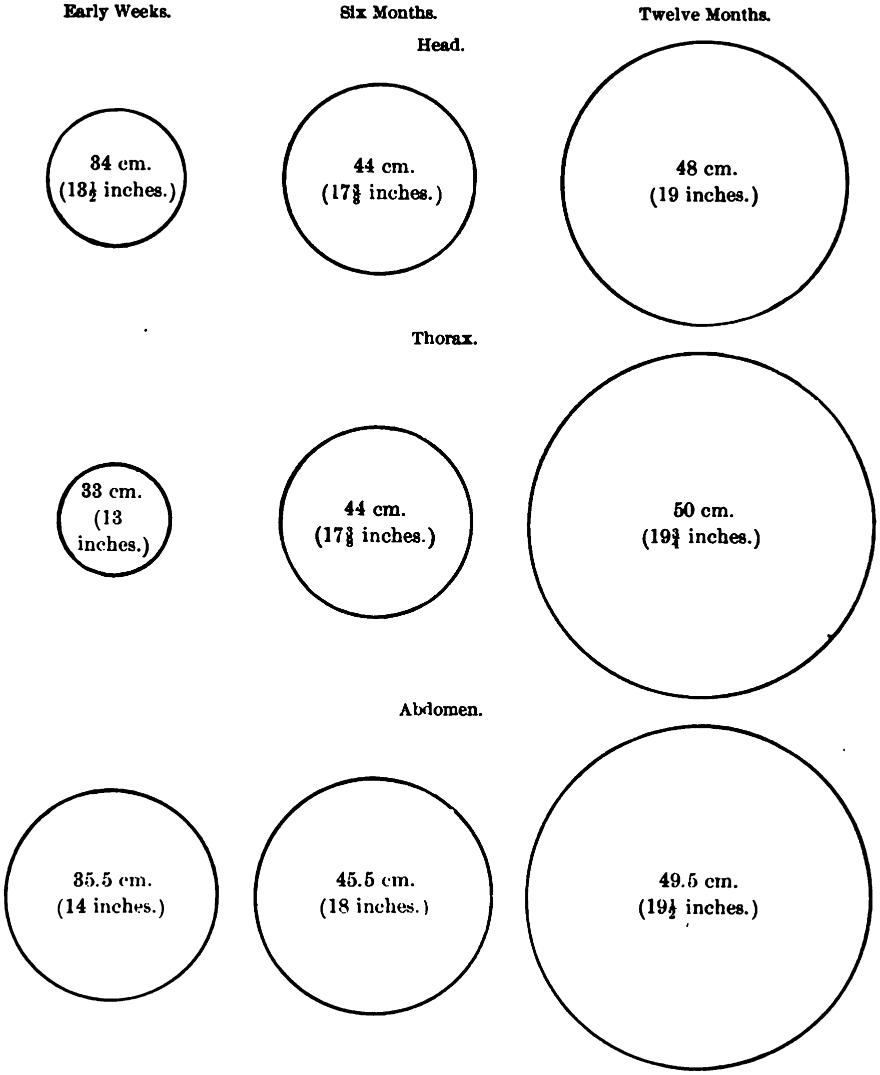
It seems rather remarkable that at ten years the distance should be as great as in the adult, but this may be accounted for by the subsequent descent of the larynx, and also, probably, by its proportionate enlargement (at least in the male) about puberty.

The peculiarities of the relations of the top of the larynx and pharynx to the spine in the young child are points of much practical importance. The changes which occur during growth depend largely on changes in the base of the skull, and on the downward growth of the jaws, which will be considered presently.

HEAD.—CIRCUMFERENCE AND CIRCUMFERENCE RELATIVE TO THORAX.— The measurement of the circumference of the head increases very rapidly, and in early childhood almost attains that of the average adult's head.

At birth the average circumference of the head is about 33 cm. (13 inches), and the thorax 1 or 2 cm. (½ or ¾ inch) less. The following table shows the relative and proportional growth of the head and thorax up to the period of puberty. These figures represent measurements from somewhat over one hundred cases. It will be seen that the circumference of the thorax has become equal to that of the head by the end of the first year; though in exceptional cases the thorax surpasses the head at a much earlier period, and I have even seen it exceed the circumference of the head at birth. Again, in some cases, the head, even in the second year, remains larger than the thorax. After the second year, the measurements of the head vary very little, and depend more upon the individual

FIG. 10.



Relative circumferences of head, thorax, and abdomen.

than upon the age; the thorax, on the contrary, increases year by year. The measurements which are given were taken almost entirely from boys. Girls, for the same age, show a proportionately smaller circumference for the thorax and also for the head.

TABLE 4.

Circumferences of Head and Thorax from Birth to Thirteen Years.

Age.	Head.	Thorax.
Birth	33 cm. (13 inches)	31 cm. (12½ inches).
5 weeks	38 cm. (15 inches)	36 cm. (14½ inches).
5 months	42 cm. (16½ inches)	41 cm. (16¼ inches).
9 months	45.5 cm. (18 inches)	43 cm. (17 inches).
1 year	45.5 cm. (18 inches)	47.5 cm. (18¾ inches).
2 years	48 cm. (19 inches)	51 cm. (20⅛ inches).
3 "	51 cm. (20⅛ inches)	55 cm. (21¾ inches).
4 "	53 cm. (21 inches)	54 cm. (21¼ inches).
5 "	53 cm. (21 inches)	54 cm. (21¾ inches).
6 "	52 cm. (20½ inches)	55 cm. (21¾ inches).
7 "	54 cm. (21¼ inches)	54 cm. (21¼ inches).
8 "	53 cm. (21 inches)	59 cm. (23⅛ inches).
9 "	54 cm. (21¼ inches)	61 cm. (24 inches).
10 "	53 cm. (21 inches)	62 cm. (24½ inches).
11 "	56 cm. (22⅛ inches)	63 cm. (24¾ inches).
12 "	53.5 cm. (21⅛ inches)	63 cm. (24¾ inches).
13 "	54 cm. (21¼ inches)	66 cm. (26 inches).

The series of circles on page 44, representing the circumferences of the head, thorax, and abdomen, shows what may be expected as to the relations of these parts of the child in the first year. They represent the average of a number of actual cases observed by me.

No especial significance need be given to the circumference of the abdomen in this connection beyond what has been previously said concerning the liver, as its measurements, of course, vary very much normally according to the degree of distention present.

Fig. 10 shows that, although there is great activity shown in the growth of the head, this activity is still greater in regard to the thorax.

FONTANELLES.—The *posterior fontanelle*, although ordinarily quite perceptible at birth, soon disappears, either from overlapping of the bone or from a permanent closure, and is usually imperceptible by the sixth week.

The *anterior fontanelle* seems to grow larger as the infant grows older, up to about the ninth month; this point is, however, disputed, and the increase may be apparent rather than real. It also seems to remain stationary, or almost so, from the ninth to the twelfth month, and then decreases slowly. It should be closed by the nineteenth to the twentieth month.

FACE AND CRANIUM.—The proportion of the face to the cranium in infancy is as 1 to 8. Froriep has also made observations on this point in older children, and finds the following proportions:

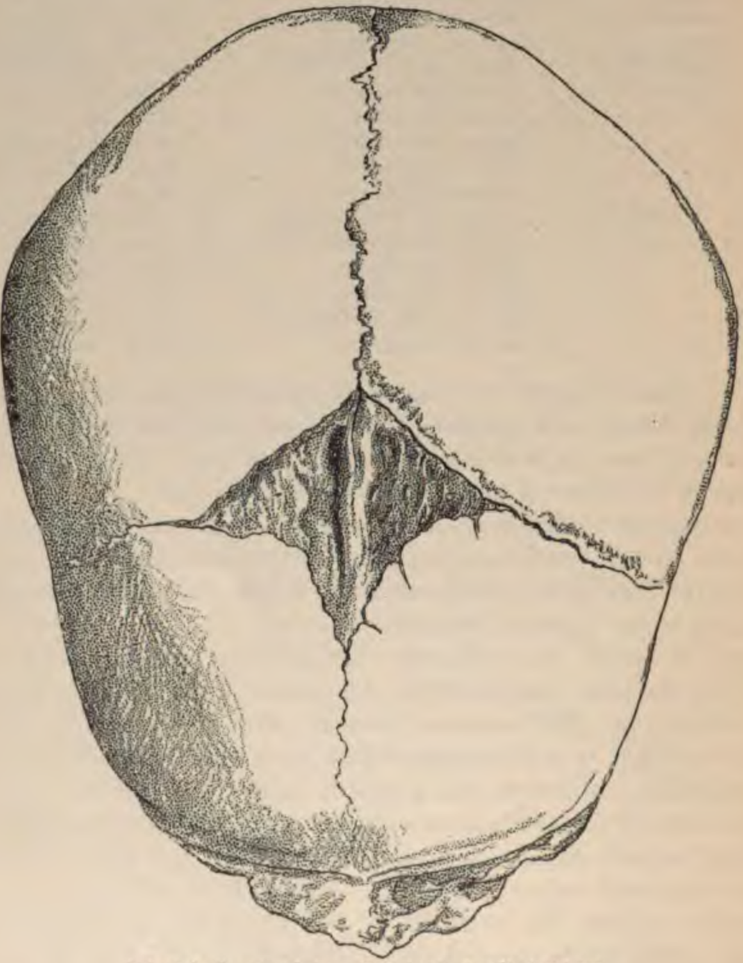
TABLE 5.

Proportions of Face to Cranium.

Age.	Face.		Cranium.
Early infancy	1	to	8
2 years	1	to	6
5 years	1	to	4
10 years	1	to	3
Adult female	1	to	2½
Adult male	1	to	2

The small size of the facial portion of the skull in infancy and early childhood is shown in Fig. 12, page 49, of the infant at birth and at three years, and also in Figs. 28 and 29, facing page 86, of the infant at birth and at nineteen months.

FIG. 11.



Infant skull, natural size. Anterior fontanelle 4 × 3 cm.
Warren Museum, Harvard University.

As the child develops, very important changes occur in the base of the skull, one of the greatest of which is the downward growth of the face. Originally the base of the skull is practically flat. The sudden rise of the basilar process in front of the foramen magnum, the angle formed with it by the body of the sphenoid, and then the sharp descent of the vomer, are adult characteristics of which at birth there is little trace. The nasal cavity is shallow and relatively long, the posterior nares are small, and the vomer approaches the horizontal. The nasopharynx has, therefore, very little height. The alveolar processes are still undeveloped, and the ramus of the lower jaw is very oblique, so that the cavity of the mouth is small. As a consequence, the larynx is placed very high up. One of the chief causes of its descent is the downward growth of the face.

BRAIN.—Up to the seventh year the brain shows an active growth, and after that year increases slowly in weight. The convolutions are not fully developed at birth, and are gradually perfected as the child grows older. The various centres of the brain which gradually become so highly developed in later childhood have but little action, so far as we can judge, at birth and in the early weeks.

Dura Mater.—An important anatomical condition in connection with the brains of young subjects is that the dura mater is much more adherent to the skull, and thus presents an obstacle to the collection of extravasations between it and the bone.

Subarachnoid Space.—The subarachnoid space usually contains a larger amount of fluid in childhood than in later life.

EAR.—The osseous meatus is not developed until about the fourth year. In introducing the aural speculum under four years of age, the ear should be drawn forward and downward instead of upward and backward as in older children and adults, or the canal will be bent on itself.

PETROSQUAMOSAL SUTURE.—The time at which the *petrosquamosal suture* closes is not at present known.

NASOPHARYNX.—According to Disse, the nasal cavity begins to increase in height directly after birth, and goes on pretty rapidly until the beginning of dentition, when it is slow until the second year is completed. After the first set of teeth are cut, the growth is rapid until the end of the seventh year. The increase in breadth occurs in the last-mentioned period, which also is the time in which the growth of the olfactory portion is most marked. Disse states that the posterior opening doubles its size in six months, remains stationary until the end of the second year, and then increases again. The following measurements were made by Dwight:

TABLE 6.

Age.	Height of Posterior Nares.	Breadth between Pterygoid Processes at Hard Palate.
About birth	6 to 7 millimetres	9 millimetres.
From 12 to 16 months	13 "	16 "
" 12 to 18 "	15 "	16 "
" 14 to 20 "	14 "	17 "
" 18 months to 3 years	15 "	21 "
" 2 to 4 years	15 "	20 "
About 6 years	16 "	20 "
7 or 8 years	20 "	22 "
About 11 years	18 "	22 "
17 years, female	22 "	20 "

These figures show that the height does not gain the predominance until adult age. At the end of the seventh month the nasal cavity approaches the adult shape, though it seems broad in proportion, and has not, of course, attained its full size. Merkol has shown that in later adolescence the growth of the respiratory portion takes place chiefly in the middle meatus. In infancy the posterior border of the vomer is very oblique. With the growth downward of the jaw this obliquity is much diminished at the age of seven or eight years.

The change in the shape of the pharynx in the first two or three years is very great, and the pharynx of older children resembles more that of the adult than that of the infant. Indeed, at four weeks we find the tip of the epiglottis on a level with the lower part of the odontoid process.

Eustachian Tubes.—The course of the Eustachian tube and the position of its opening undergo changes corresponding to the development of the nasal cavity. At birth the tube is horizontal, or nearly so. In the adult the cartilaginous portion slants downward. Nevertheless, the opening of the tube is opposite a higher part of the nose in the adult than in the child. In the fœtus the opening is below the level of the hard palate, which it reaches at birth. Up to the ninth month after birth, according to Disse, there is but little change. After that time, however, the opening is distinctly higher than the floor of the nasal cavities. At four years, Kunkel found it to be three or four millimetres higher. In the adult it is opposite the end of the inferior turbinate bone.

Pharyngeal Tonsil.—The pharyngeal tonsil increases after birth, and by the end of the first year has a length of eighteen millimetres.

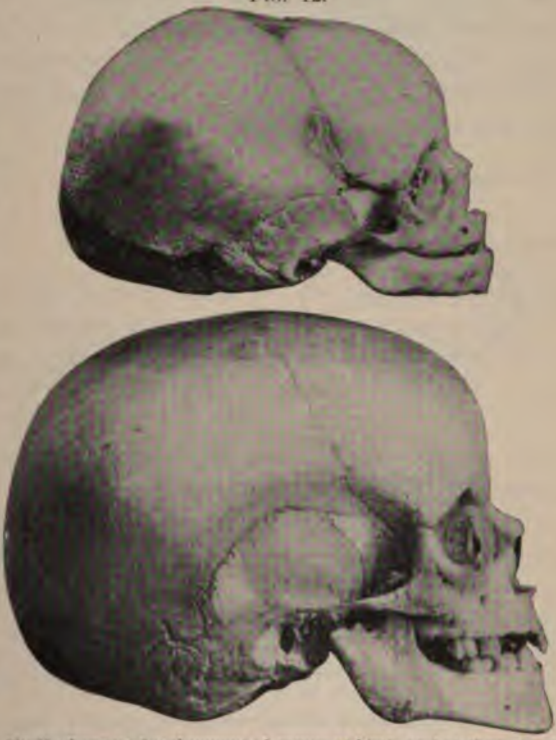
HARD PALATE.—In a child of three years or less the line of the hard palate strikes about the middle of the basi-occipital bone. It would hardly be possible, without passing the finger round the soft palate, to feel much higher than the arch of the atlas. The base of the odontoid process would be under the mucous membrane seen at the back of the throat through the open mouth. The tip of the epiglottis is at the junction of the odontoid with the body of the axis. Only the very top of the third vertebra could be satisfactorily explored. At six and at thirteen (Symington's plates) the line of the hard palate has about the adult direction,—that is, it strikes about the top of the atlas or the basilar process near its beginning. In both the finger could probably examine the vertebræ from the first to the fourth inclusive. The atlas, however, would be reached with much more difficulty in the older than in the younger subject, as the relations oft he soft palate are more nearly those of the adult.

MOUTH.—As the infant grows older the mouth becomes an organ more adapted for certain uses beyond that of a mere means of entry for the food to the stomach.

MAXILLARY BONES.—The ossification of the maxillary bones begins early, progresses slowly, and, together with the final formation of the jaw, is completed at puberty. Fig. 12 represents the characteristic incomplete development of the ramus of the inferior maxillary bone in the early weeks and months of life, and its almost complete development at three years.

The chief characteristic, as seen in Fig. 12, is the oblique angle which the ramus makes with the body of the bone at birth, and this becomes more evident when compared with the jaw at three years. The much greater proportion of the ramus to the body of the bone at three years, and the nearer approach to a right angle where they join are noticeable.

FIG. 12.



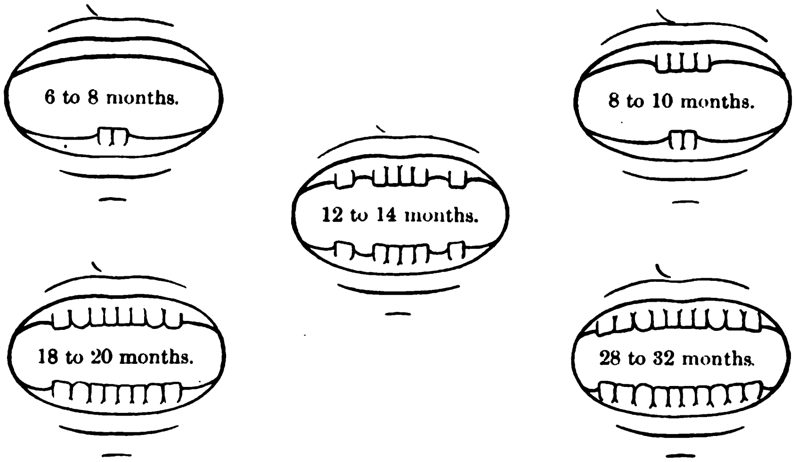
Skulls showing development of ramus at birth and at three years. Warren Museum, Harvard University.

Teeth.—The development of the first set of teeth begins at about the seventh week of intra-uterine life, and, progressing slowly, is completed about the end of infancy. At birth the twenty embryo teeth, ten in each jaw, are so enclosed in the alveolar processes that nothing but the smooth mucous membrane is apparent on the gums above. Below, they are connected with the branches of the inferior dental nerve (an important clinical fact to be remembered) through openings at the bottom of the alveolar processes. When calcification of the neck of the tooth begins, elongation also takes place, and, as the tooth is enclosed in bony walls below and on the sides, it gradually grows through the point of least resistance, namely, the gum, which covers the top of the alveolar processes. The continued pressure gradually causes atrophy of the mucous membrane, and the crown of the tooth appears on the edge of the gums. The various teeth come through the gum at times which are regulated according to their development, that is, at times corresponding to the calcification of their roots and consequent elongation. This process usually takes place in groups and with considerable regularity in the average normal infant. Variations, both as to the order in which the teeth appear and in the time of their appearance, are so common that it seems hardly practicable to have set rules designating these times. The experience of different physicians seems to differ, but all practically are guided by very general rules. An infant may be born with one or more teeth.

LANE LIBRARY

The first tooth may appear at any time during the first year of life, or may be delayed until the second year without any other apparent vice of development. In like manner, every kind of variation may be met with in the order in which the teeth appear, without the slightest evidence of any pathological condition, mental or otherwise, being found either at the time or later. It is therefore unnecessary to alarm the parents by stating that their child is abnormal because it has not cut a tooth in the first year. We should, however, carefully watch these children and be sure that their food contains the proper nutritive elements not only for their age, but also for their individual digestion.

FIG. 18.



Five periods of development in the first dentition.

The appearance of the teeth in groups suggests certain practical divisions to determine various questions, such as the best time for weaning, or for vaccination. These divisions constitute the *dental* and *interdental periods.* In my individual experience, the first tooth appears at about the sixth or seventh month, though at times I find it much earlier, as at the fourth month, and later, as at the ninth, tenth, eleventh, or twelfth month. The first tooth which develops sufficiently to come through the gum is in most cases one of the middle lower incisors. The groups and the dental periods, allowing always for many variations, are as follows:

TABLE 7.

Temporary Teeth. First Dentition. Twenty in Number.

	Dental Periods.	Groups of Teeth.
I.	6 to 8 months	2 middle lower incisors.
II.	8 to 10 months	4 upper incisors.
III.	12 to 14 months	2 lateral lower incisors and 4 first molars.
IV.	18 to 20 months	4 canines.
V.	28 to 32 months	4 second molars.
		20

The second set of teeth begins to replace the first at about the sixth year.

TABLE 8.

Permanent Teeth. Second Dentition. Thirty-two in Number.

Years.	Groups.
6	4 first molars.
7	4 middle incisors.
8	4 lateral incisors.
9	4 first bicuspids.
10	4 second bicuspids.
11	4 canines.
12	4 second molars.
17 to 25	4 third molars (wisdom-teeth).
	32

The first four teeth of the second dentition are usually called the sixth-year molars. They do not replace any of the permanent teeth, but, the jaw having grown so as to provide space back of the temporary teeth, they appear back of and next to the second molars. This usually occurs at about the sixth year.

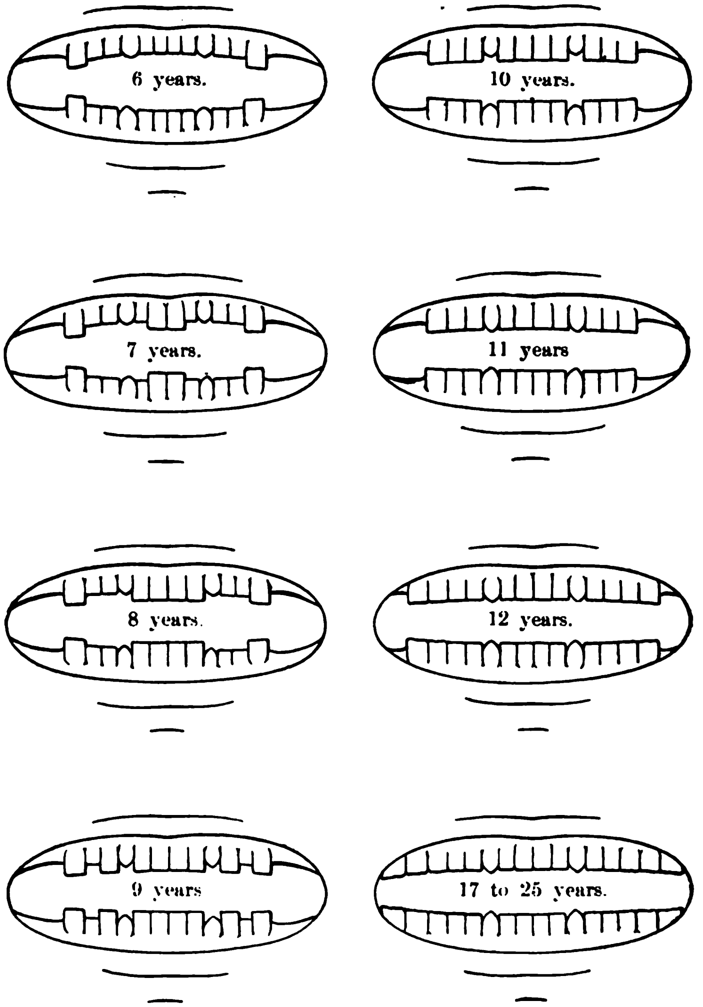
In the seventh and eighth years the permanent incisors replace those of the temporary set. In the ninth and tenth years the bicuspids replace the temporary molars. In the eleventh year the permanent canines replace the temporary, and in the twelfth year the four second molars appear. This really completes the second dentition of childhood, twenty-eight teeth. The remaining four molars belong to a period of adult growth. Fig. 14 (page 52) shows the manner in which the permanent teeth replace the temporary set between the ages of six and twenty-five years.

THORAX.—Top of Sternum.—From the few observations which have been made, the top of the sternum in infancy and early childhood seems to be opposite some part of the first or second dorsal vertebra. (Dwight.)

Diameters.—The anteroposterior diameter of the interior of the thorax is to the transverse diameter at three years, according to Dwight's "Frozen Sections," as one to two, and in a child of from five to six (Symington) the depth is even relatively greater. The ribs bend much less backward than in the adult, and the back first becomes rounder and then flatter. At four or five years great progress in growth has been made, and the infantile form of the thorax has wholly disappeared. Slight changes, however, probably go on for some years.

Ossification.—Towards the end of the first year the bone-centres of the sternum have grown, and the sternum has gained a good deal in stability. New points of ossification have probably appeared, but still the sternum is essentially cartilaginous, the bone merely consisting of islands in a sea of cartilage. At two years of age the manubrium and the second and third pieces are nearly ossified, but their shape is made by their cartilaginous borders. At three years I have twice seen the manubrium and the second piece of the sternum presenting in bone their real shape, while the third piece was still framed in cartilage. Sometimes, however, the process of ossification is more backward. The ossification of the lower part of the sternum is less advanced than the upper part. As to its relative size opinions differ. Probably the individual variation is very great. The ribs being comparatively horizontal, the cartilages rise very little, and at the lower part of the chest in front they are nearly together, making narrow intercostal spaces, the seventh cartilages often meeting below the body of the sternum. In the dead body of a young child, especially if it

FIG. 14.

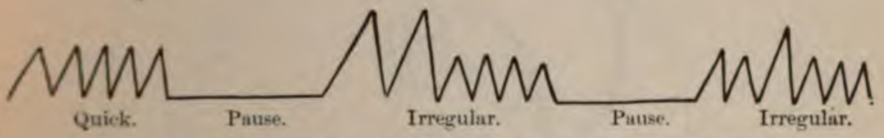


Eight periods of development in the second dentition.

be emaciated, it is striking to see how, after the cadaveric rigidity has passed away, the sternum and cartilages, forming the front of the chest, fall in at the point where they join the ribs.

RESPIRATION.—At birth there is no decided type of respiration for the two sexes. As the infants, both male and female, however, grow older and a more equable respiratory mechanism becomes established, I have found that, as a rule, in the early months of life, the type of respiration is abdominal. Fig. 15 shows the irregular respiration of infancy.

FIG. 15.



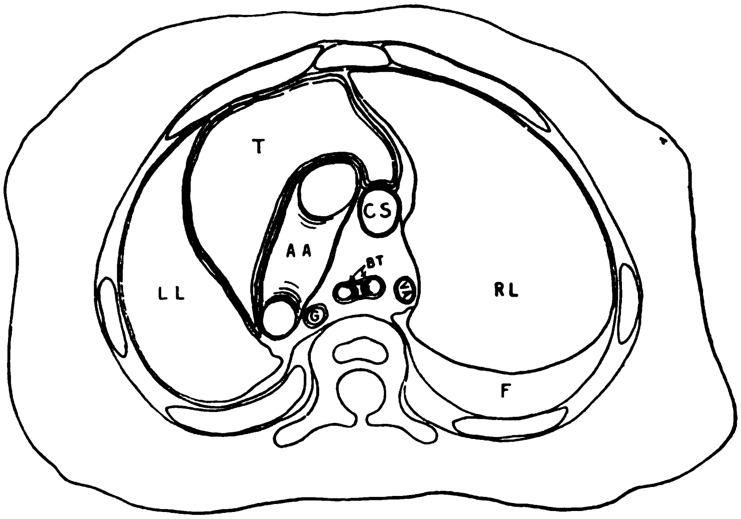
Respiration for one-half minute in a healthy infant nine months old; awake, but quiet.

DIAPHRAGM.—A limited number of observations by Dwight showed the diaphragm to be opposite some part of a space between the lower part of the eighth and the upper part of the tenth vertebra.

THYMUS GLAND.—The *thymus* is most developed in the first two years of life, but it persists longer than was formerly taught. During its greatest development it is found in the neck as well as in the thorax, extending perhaps 2 cm. ($\frac{5}{8}$ inch) above the sternum, which, you must remember, is no small part of the surface of a child's neck. The thymus extends down the anterior mediastinum, lying on the pericardium in two long lobes on either side of the median line. The extent of these lobes is very variable, and the two are not usually symmetrical. I have seen them, even in an infant, so developed that the longer nearly reached the lower end of the sternum; but it is very uncommon for it to reach the diaphragm. These prolongations become thinner as they descend. The thymus is a thick mass behind the first piece of the sternum, where it rests on the top of the heart against the great vessels concealing the innominate veins, more or less of the superior vena cava and the arch of the aorta, and extending back to the trachea. Lower down it extends on either side into the angle between the pericardium and the lungs, or rather pleuræ. A section of the thymus made by Dwight was found to run nearly horizontally from the top of the fourth dorsal vertebra to just above the junction of the second costal cartilage with the sternum.

The cavity of the thorax seems to be divided into three parts, one on either side of the lungs and a median one occupied by the thymus, the transverse part of the arch of the aorta, with the superior vena cava on its right, and the trachea and œsophagus behind. The area occupied by the thymus is very nearly equal to that of the left lung. The thymus reaches backward on the left of the aorta behind the level of the front of the spinal column. There is also what seems to be a piece of it between the vena cava and the trachea. On the upper surface of the same section, at about the level of the sternoclavicular articulations, it is in front of both innominate veins and behind the right one. The lungs are prevented from approaching each other so nearly behind the manubrium as they do in the adult.

FIG. 16.



Frozen section, child of three years: RL marks right lung; LL marks left lung; T marks thymus gland; G marks gullet; CS marks superior vena cava; AA marks aortic arch; VA marks vena azygos; F marks some fluid which happened to be in the right chest; BT marks bifurcation of trachea.

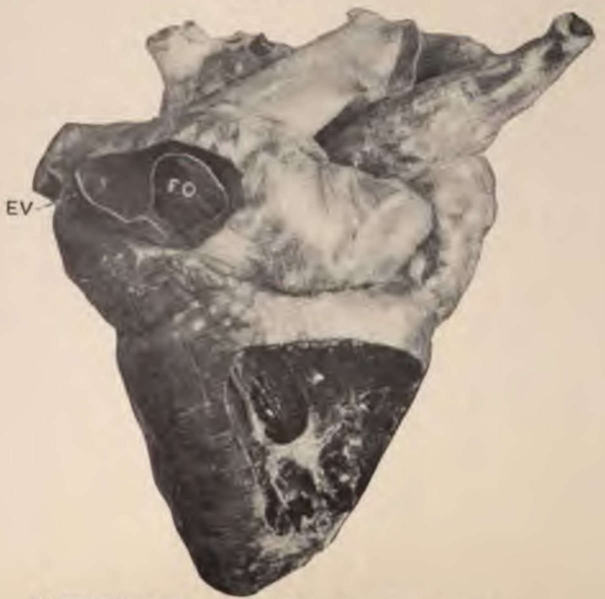
A frozen section (Dwight) of a child three years old (Fig. 16) shows that behind the manubrium there is much more of the thymus to the left than to the right of the median line, and its dulness on percussion must have been evident at the left of the sternum. Below it merges into the cardiac dulness, and no distinction between the thymus and heart is possible by percussion.

The theoretical results of enlargement of the thymus are very serious. Resting on the anterior and weaker ventricle, which is prolonged upward into the pulmonary artery, it may interfere with the pulmonary supply of blood, and by compressing the innominate veins and the superior cava it may interrupt the return of venous blood to the lungs. Whether or not it may compress the thoracic duct is doubtful, but it certainly may press on the trachea.

The thymus is said often to persist for several years after puberty, but observations are not numerous on this point. It seems to disappear from the neck and from the front of the heart and to remain longest behind the first piece of the sternum.

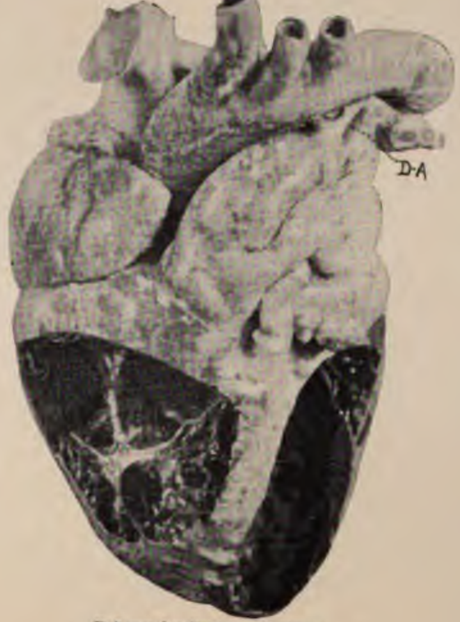
HEART.—The changes which take place in the infant's heart after birth are not simultaneous, which is a point to be remembered in making a differential diagnosis of cardiac disease during the first ten days of infancy. The following table states the time at which these changes should occur:

FIG. 17.



EV marks Eustachian valve; FO marks foramen ovale.

FIG. 18.



D A marks ductus arteriosus.

TABLE 9.

POST-NATAL CHANGES OF FŒTAL CONDITIONS.

Ductus Venosus.—The ductus venosus becomes a fibrous cord in the fissure of the ductus venosus in from two to five days.

Eustachian Valve.—The intra-uterine function of the Eustachian valve practically disappears at once at birth, but its remains can be found for an indefinite period. (See heart dissected by Dr. F. Dexter, Fig. 17, facing page 54.)

Foramen Ovale.—The foramen ovale usually closes about the tenth day, but the upper part sometimes never closes. The closed foramen ovale is seen in this same heart. (Fig. 17, facing page 54.)

Ductus Arteriosus.—The ductus arteriosus is about 1.5 cm. (¾ inch) long, has a diameter of about .25 cm. (⅛ inch), and is usually, so far as being pervious to the blood is concerned, obliterated in from four to ten days. Its remains forms a fibrous cord connecting the pulmonary artery and the aorta. (See Fig. 18, facing page 54.)

Umbilical Vein.—The umbilical vein becomes the round ligament of the liver, and is obliterated in from two to five days. As pointed out by Jacobi, it differs from the arteries very much less than is usual with the veins and arteries in other parts of the body. Its muscular layer is very large and strong.

Umbilical Arteries.—The umbilical arteries in their upper parts become obliterated in from two to five days, forming the anterior true ligaments of the bladder, while the lower parts remain pervious and form the superior vesical arteries. The umbilical arteries are usually thick and strong, owing to the great development of their muscular layer.

It is generally held that in the first year of life the long axis of the heart is more nearly horizontal than later. The apex is thought by many to be higher. This is true in the first few years, but somewhat later it may be found in the adult position, or, in cases where the lower part of the sternum is backward in development and the cartilages crowded together, it may be in a lower space than normal. It is not unlikely that a subsequent change in these portions of the walls would correct this. Thus, if in the early condition the apex were at the sixth intercostal space, a lengthening out of the lower end of the sternum might cause such a descent of the ribs as would bring it into the fifth space.

Weight.—As shown by Boyd, the weight of the heart in proportion to that of the whole body does not vary much at different ages, so that the relative labor of the heart does not materially differ between the young subject and the adult. In the first few years, however, the increase of the weight of the heart is greater than at about the fourth or fifth year, and this increase is again greater at about puberty. These are facts of practical importance to be remembered when we are studying the diseased conditions of the heart.

TABLE 10.

Weights of the Heart during its Development. (*Boyd.*)

Age.	Grammes.
At birth	20.6
1½ years	44.5
3 years	60.2
5½ years	72.8
10½ years	122.6
17 years	233.7

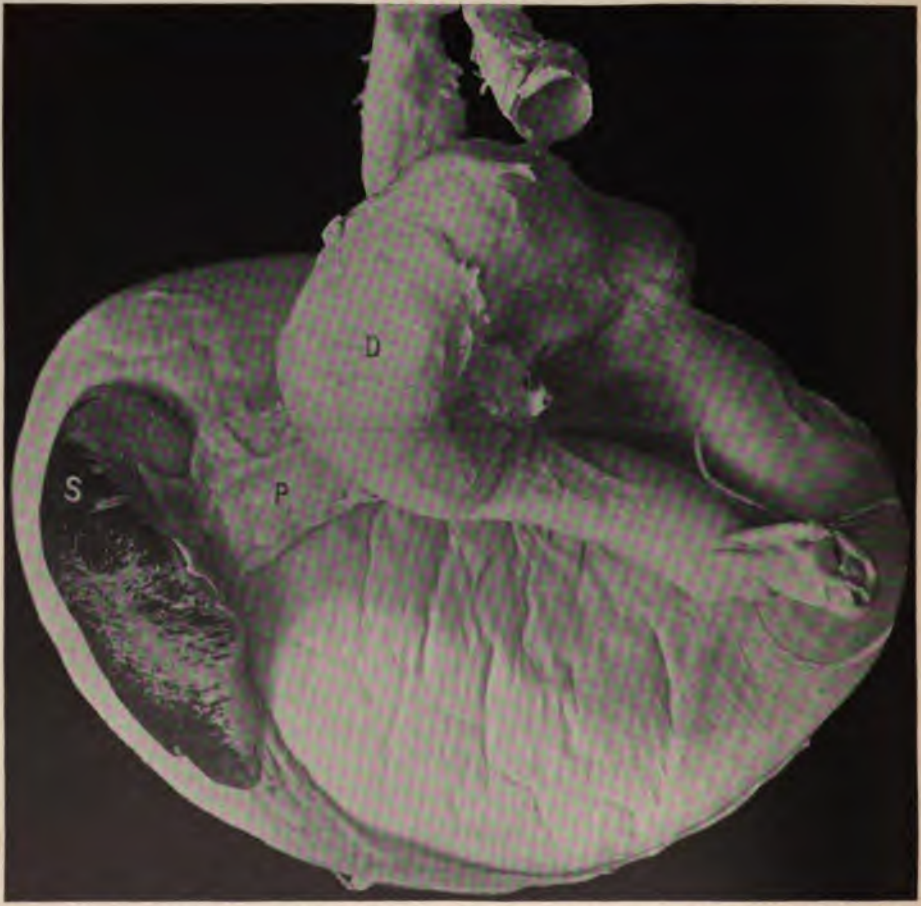
Figs. 17 and 18 show the heart in the early days of life. (Dexter.)

BLOOD-VESSELS.—"According to a number of actual observations made by R. Thoma, the post-fœtal growth is relatively smallest in the common carotid, and largest in the renal and femoral arteries. Between these two extremes there are found the subclavian, aorta, and pulmonary arteries. These are differences which correspond with the differences in the growth of the several parts of the body supplied by those blood-vessels. In regard to the renal artery and the kidney, it has been found that the transverse section of the former grows more rapidly than the volume and weight of the latter. Thus, it ought to be expected that congestive and inflammatory processes in the renal tissue were almost predestined by this disproportion between the size of the artery and the condition of the tissue. Moreover, the resistance to the arterial current offered by the kidney-substance depends also upon the readiness with which the current is permitted to pass the capillaries. It has been found experimentally that within a given time more water proportionately can be squeezed through them in the adult than in the child. These anatomical differences may therefore be the reason why renal diseases are so much more frequent in infancy and childhood from all causes, with the exception of that one which is reserved for the last decades of natural life, atheromatous degeneration." (Jacobi.)

Pulmonary Artery.—It is doubtful whether there is any essential difference at different ages in the origin of the pulmonary artery. If we say that in the infant it is rather higher than later, we have stated about all that is justifiable.

LUNGS.—At what age the lungs reach their full expansion forward has not been determined. It would appear that it is not before five or six years, and it is probably still later. As the chest expands laterally the lungs of course increase, and the relatively greater size of the heart to the lung in the infant depends essentially on the size of the lungs. During the first year of life (according to Northrup) the alveolar walls are thick and their blood-vessels are held loosely. It is not until the fourth or fifth year that the proportionate adult development between the alveoli and the bronchi is attained, and the stroma has become dense and binding, restraining the capillaries as in adult life. In infant life the underlying loose tissue lining the bronchial tubes gradually binds the mucous membrane to the fibromuscular wall. From this time it keeps pace in its growth with the other compact tissues, until in adult life it appears as dense fibrous bands. During the first two years the air-cells have not attained the proportionate capacity which exists in adult life, and the bronchial tree is still large in proportion to the dilating and multiplying alveoli. Again the air-spaces developed from the terminal bronchi have covered themselves with a continuous layer of flat nucleated epithelium. In its subsequent growth in adult life it is believed that the expanding alveolus does not increase its number of epithelial cells to cover the more extended wall, but somewhat enlarges their size, and, still further, that

FIG. 19.



Stomach, spleen, and pancreas at 10 months. Natural size, posterior view. S marks the spleen; P marks the pancreas; D, the duodenum. Warren Museum, Harvard University.

some of the flattened epithelium loses its nuclei and expands to form large, very thin plates, called respiratory epithelium.

ABDOMEN.—LIVER.—The liver is proportionately large at birth and in early childhood, and can be felt below the edge of the ribs in the right hypochondrium, its border being about 1 or 2 cm. ($\frac{3}{8}$ to $\frac{5}{8}$ inch) below the lower rib.

Gall-Bladder.—The fundus of the *gall-bladder* is in relation to the surface of the body about that of the ninth costal cartilage near the border of the right rectus muscle. (McClellan.)

SPLEEN.—There is nothing especially to be noted in the spleen in childhood, as it corresponds in its position to that of the adult. According to Foster, the spleen grows rapidly in early infancy, but in proportion to that of the adult is both absolutely and relatively smaller. It is said that the spleen when enlarged encroaches more upon the thoracic cavity than in the adult, owing to the greater resistance offered by the costocolic fold of the peritoneum upon which it rests. In many cases of enlarged spleen from varied causes which I have met in infants it has seemed to me that the abdomen was encroached upon to a greater extent than in adults, and that both the physical and the rational signs of the enlarged spleen in the thorax were relatively insignificant and often difficult to detect.

PANCREAS.—The function and the anatomy of the pancreas correspond very closely to that of the salivary glands. It is situated in front of the first lumbar vertebra, behind the stomach, and, according to the variations produced by age and the growth of other parts, lies somewhere between the umbilicus and the ensiform cartilage.

The relative position of the spleen and pancreas to the stomach and duodenum is shown in Fig. 19. The organs were obtained at the autopsy of an infant ten months old. The spleen is behind the cardiac end of the stomach, and very near its extremity. The pancreas extends from the spleen (its tail being in close apposition to the latter organ) along the posterior surface of the stomach and somewhat upward to the smaller curvature, passing behind the duodenum and its head resting in the concavity of the duodenum. The curve of the duodenum is also clearly shown in the specimen.

KIDNEYS.—The kidneys are lobulated at birth. This condition continues for a long time and then disappears, the lobulation being represented by the pyramids of Malpighi. A few years after birth the position and relations of the kidney approximate those of the adult (McClellan).

Suprarenal Capsules.—The suprarenal capsules are relatively large in size, and gradually approach the adult proportions as the child grows older.

BLADDER.—Although small at birth, the bladder soon becomes capable of great distention.

Symington, from a frozen section which he made in the median plane through the body of a child seven months old, shows the position of the bladder, which happened to be distended. It takes up, practically, the whole of the lower portion of the abdomen, an observation which at once suggests the difficulty of making a correct physical examination of the infantile abdomen during life, unless certain that the bladder is empty.

The above fact was strikingly exemplified in a little girl, three years old, who was in my wards at the Children's Hospital. She was sent to the hospital for an examination in reference to the advisability of an operation to remove an abdominal tumor. On inspection, a rounded prominence extending from the pubes to 3 cm. (1¼ inches) above the umbilicus could be plainly seen. By palpation the tumor could be felt extending from the right inguinal region over to the crest of the left ilium. The tumor was soft, elastic, and fluctuating. It was evidently not in the abdominal walls, but intra-abdominal. The child was said to have been ailing for over a week, and to have grown thin. She passed her urine frequently, but in small quantities. Nothing abnormal was found on an analysis of the urine.

Suspecting a distended bladder, a catheter was introduced, and 270 c.c. (9 ounces) of urine were withdrawn. The tumor immediately disappeared, and the abdomen became soft and resonant.

A practical lesson to be drawn from this case is, that the bladder should invariably be carefully examined and emptied before diagnosticating or operating in abdominal disease. I have seen a distinguished laparotomist neglect this precaution in a young child while operating for appendicitis, and on opening the abdominal cavity cut directly through the walls of the bladder. The urine flowing out through the wound was the first indication to him that he had failed to appreciate that in early life the bladder is essentially an abdominal organ.

STOMACH.—Many peculiarities of the digestive tract may arise from such causes as the different proportionate stages of development of the parts of the gastro-enteric tract at different ages, and to differences in their peritoneal attachments. The stomach grows very rapidly, and peculiarities of shape appear at an early age. I have seen a stomach of four and one-half months which, although small, was relatively broader than in the adult. The adult shape, however, is soon acquired. How permanent this may be is as yet unsettled. There is no doubt that great dilatation may be induced, and it is highly probable that where too small quantities of food are given the normal stomach will contract. It is also very likely that certain shapes are acquired at a very early period. Dwight has shown me in a young child a well-marked *antrum pylori*,—that is, a pouch above the pylorus, which, in extreme cases, forms almost a separate chamber. It is evident that the clinical significance of our anatomical knowledge of the growth of the stomach in the first year is very great. This question of growth is, in fact, one of the most important factors in the problem of the substitute feeding of infants, and a lack of its thorough comprehension often leads to most unfortunate results.

Capacity.—There has been much dispute as to the proper method of determining the gastric capacity during infancy. All methods are open to criticism, but by combining all the methods and making general deductions we can arrive at practical conclusions concerning the size of the stomach at different ages. One of the methods which may be employed is a clinical one, and is as follows:

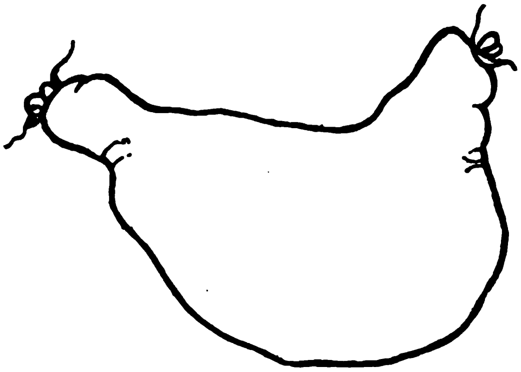
A mother is selected who is young, healthy, has plenty of good milk, and who has a healthy infant of normal size and weight, and appetite according to its age, and one that is thriving,—that is, digesting well and gaining regularly about 30 grammes (1 ounce) a day. The infant is first carefully weighed; next it is allowed to nurse until it is evidently satisfied,—that is, practically, until it feels that its stomach is full. The infant is then weighed again, and the increase in weight denotes the gastric capacity in ounces of milk. Careful investigation has shown that one fluid ounce of milk weighs very nearly 30 grammes. Certain control experiments have shown that this method of determining the gastric capacity is a practical one. It is well known among those who deal in cattle that when fat cattle are transported long distances, as from Chicago to New York, they are found to have lost materially in weight, perhaps thirty or forty pounds. If these cattle are allowed to fill their stomachs with water, an increase in their weight will be found corresponding exactly to the weight of the water which they have drunk.

If a number of healthy infants of different ages and of average weights are fed and weighed in this way, we can approximately, by comparing the gains in weight which correspond to the same ages, determine the gastric capacity for each age. I should not, however, consider this by itself a reliable method for determining the gastric capacity, as it is open to many objections, which need not be discussed at present. One source of error, for instance, is the variation of the infant's appetite, which may cause either too great distention or underfilling of its stomach. Another method is the actual measurement of the gastric capacity at the autopsy, with suitable precautions to avoid over-distention. In this way it has been determined that the stomach grows very rapidly in the first three months after birth, grows slowly in the fourth month, and is then almost quiescent for about two months. It then begins to grow again until it has reached its adult size. Frolowsky's rules for determining the gastric capacity of young infants approximate in their results so closely my own investigations that I have prepared from them figures of infants' stomachs at different ages and at different periods of growth. The tracings of the stomachs are life-size. Frolowsky shows that the activity of the stomach's growth is very great in the first quarter of the first year, that it is very slight in the second quarter, and that it again shows a moderate activity in the last part of the year. He represents this activity of the stomach's growth by the ratio of 1 for the first week to $2\frac{1}{2}$ for the fourth week and $3\frac{1}{5}$ for the eighth week, while it is only $3\frac{1}{3}$ for the twelfth week, $3\frac{4}{7}$ for the sixteenth week, and $3\frac{3}{5}$ for the twentieth week. As a starting-point from which to calculate the above proportions I have taken the infant's stomach shown on page 34, Fig. 8, which is also intended to represent an infant of the average birth weight.

FIG. 20.

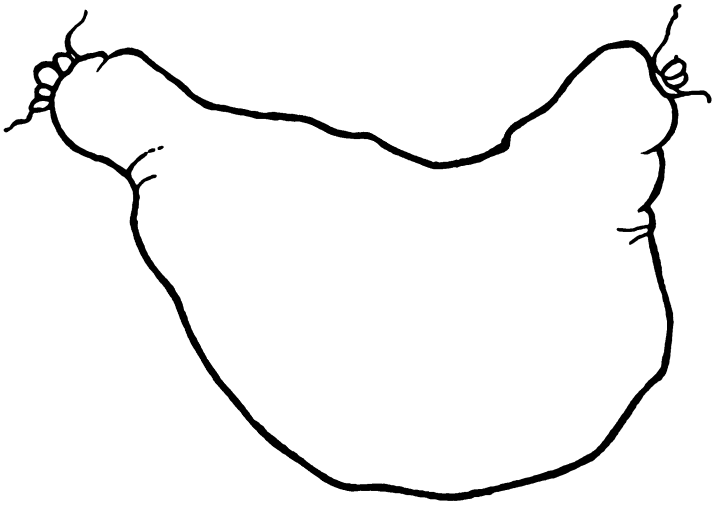
Gastric Capacity in the First Five Months of Life.

I.



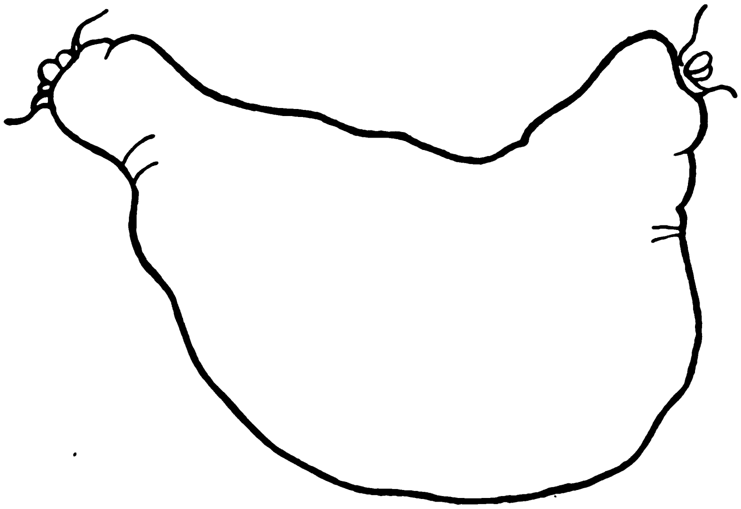
Infant 3 hours old. Capacity of stomach, 25 to 30 c.c. (⅚ to 1 ounce).

II.



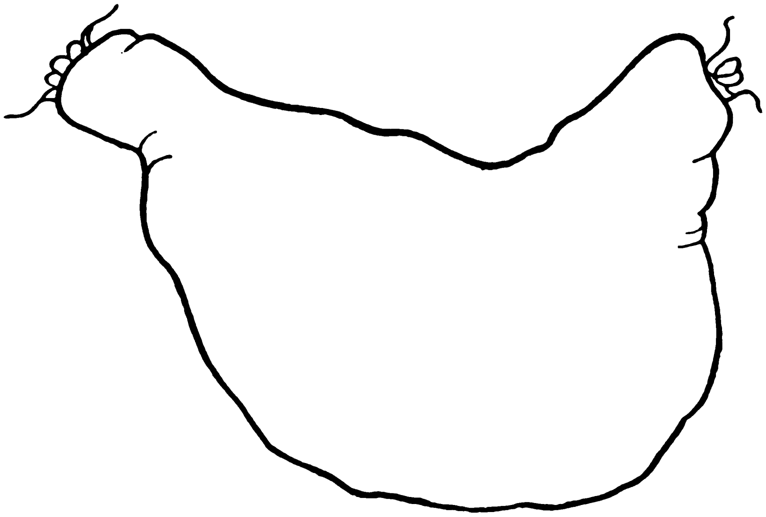
Infant 4 weeks old. Stomach 2½ times larger than I. Capacity, 75 c.c. (2½ ounces).

III.



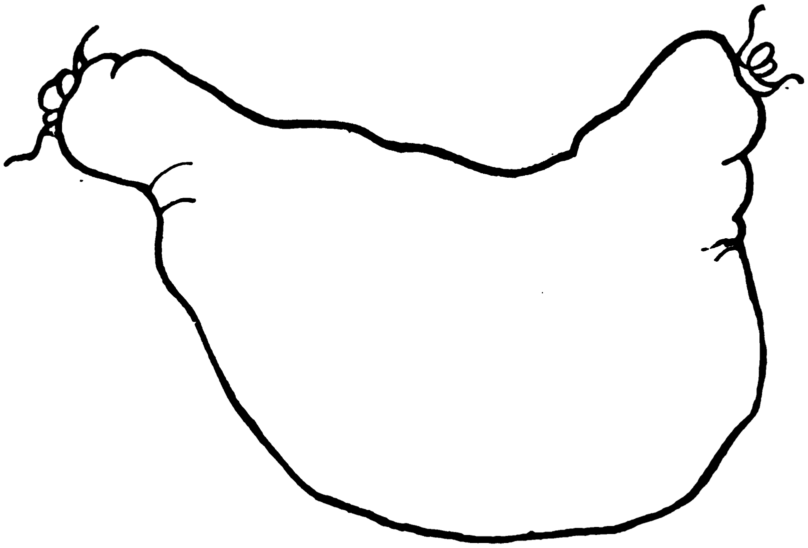
Infant 8 weeks old. Stomach 3¼ times larger than I. Capacity, 96 c.c. (3¼ ounces).

IV.



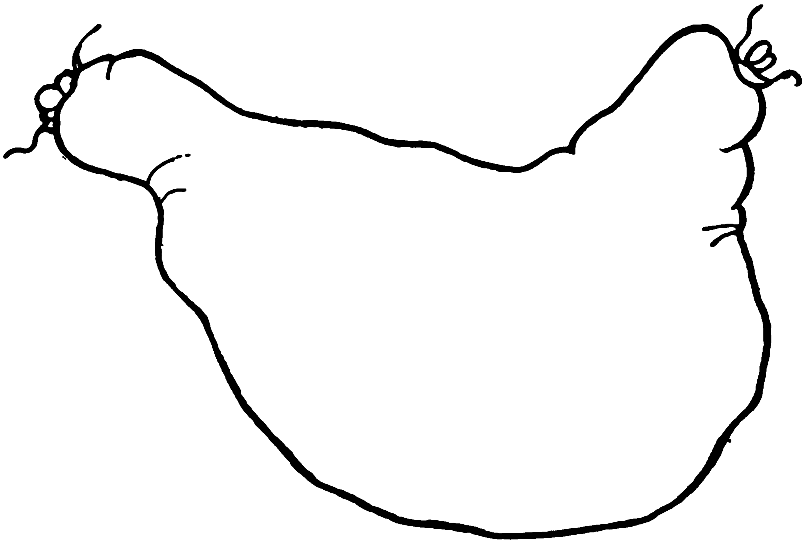
Infant 12 weeks old. Stomach 3⅓ times larger than I. Capacity, 100 c.c (3⅓ ounces)

V.



Infant 16 weeks old. Stomach 3¼ times larger than I. Capacity, 107 c.c. (3.56 ounces).

VI.



Infant 20 weeks old. Stomach 3⅓ times larger than I. Capacity, 108 c.c. (3.6 ounces).

In comparing these measurements with actual stomachs, the gastric capacity as given for sixteen and twenty weeks is somewhat small. They are, however, extremely valuable to begin with, as it is always better to err on the side of giving too little food than too much.

The gastric capacity, according to Fleischmann, is greater at the same age in the artificially fed than in the breast-fed infant. This observation, however, in all probability only emphasizes the importance of bearing in mind the normal gastric capacity of the different ages, and of using this knowledge to prevent the overfeeding which has produced so noticeable a difference between the sizes of the stomach in breast-fed and artificially fed infants.

Since the development of the methods of substitute-feeding in connection with milk modification, there is no reason to suppose that when the infant's stomach has been properly managed it is any larger than in breast-fed infants.

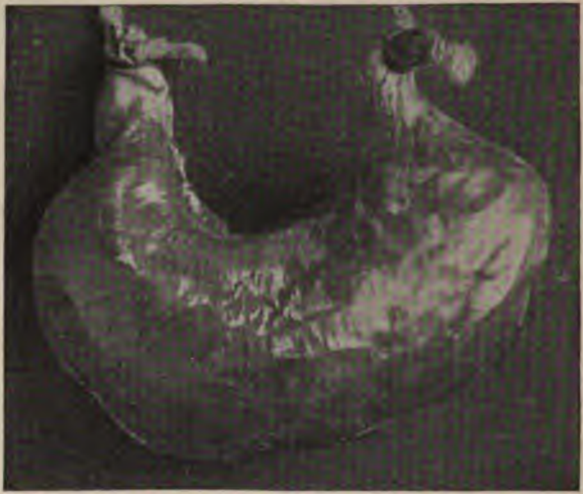
The cause, however, which produces the most uniform individual difference in the gastric capacity at the same age is the weight of the infant. In general it can be said, the greater the weight the greater the gastric capacity. A good illustration of the correctness of this rule has lately come under my notice, where a breast-fed infant of twelve months with a stomach normal in shape presented a gastric capacity of only 90 to 105 c.c. (3 to 3½ ounces). This capacity corresponded to its weight, 4289 grammes (9½ pounds), about the average normal weight of an infant at eight or ten weeks, rather than to its age, which in the average infant would present a gastric capacity of 240 c.c. (8 ounces).

I have also had under my care an infant of six weeks whose general development and weight corresponded so closely to those of the normal average infant of twelve weeks that it was self-evident that the two ounces of food which would ordinarily have been the proper allowance, so far as the age was concerned, was not sufficient, and that its weight indicated a gastric capacity for an allowance of four ounces, which, in fact, it took and digested with the greatest ease, while with any amount less than the four ounces it was never satisfied.

In this connection it is important to note that at autopsies in measuring the gastric capacity it should be done before the stomach is separated from its mesenteric attachment, as otherwise it is easily stretched by the fluid introduced, and will then show a greater capacity than would be within the normal limits during life. In looking at the following illustrations, which represent the actual sizes of a number of stomachs which have come under my observation, it is interesting also to note the different shapes which these stomachs represent, as some of them are very different from the usual classical figures represented in books. So far as could be ascertained, these shapes did not occur from any especial disease, such as would influence the outline of the stomach.

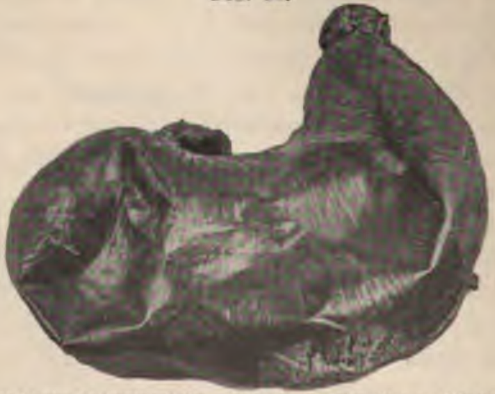
On examining Fig. 8 (page 34) and Figs. 21 and 22, it is clear that one cannot always depend on an infant's weight for determining its gastric capacity in the early days of life. Thus, the weights of 2500 grammes, 3000 grammes, and 4000 grammes all had the same gastric capacity of 25 c.c. Again, the weight of 2700 grammes had a greater capacity, 40 c.c., than the weights of 3000 and 4000 grammes. One must, however, also allow that there might be an error in measuring the gastric capacity.

FIG. 21.



Stomach of infant 2½ days old, natural size. Gastric capacity, 25 c.c. Weight, 4000 grammes.

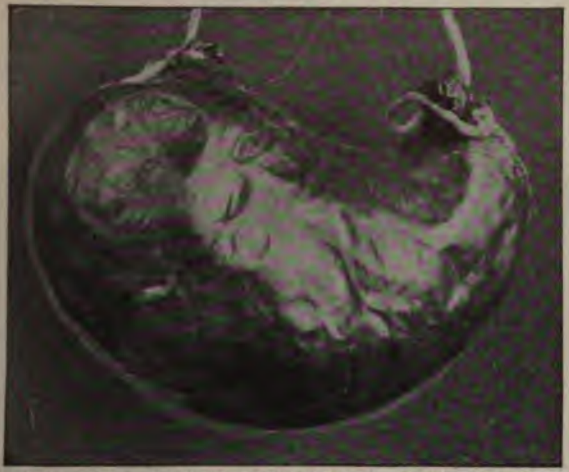
FIG. 22.



Stomach of Infant 5 days old, natural size. Gastric capacity, 25 c.c. Weight, 3000 grammes.

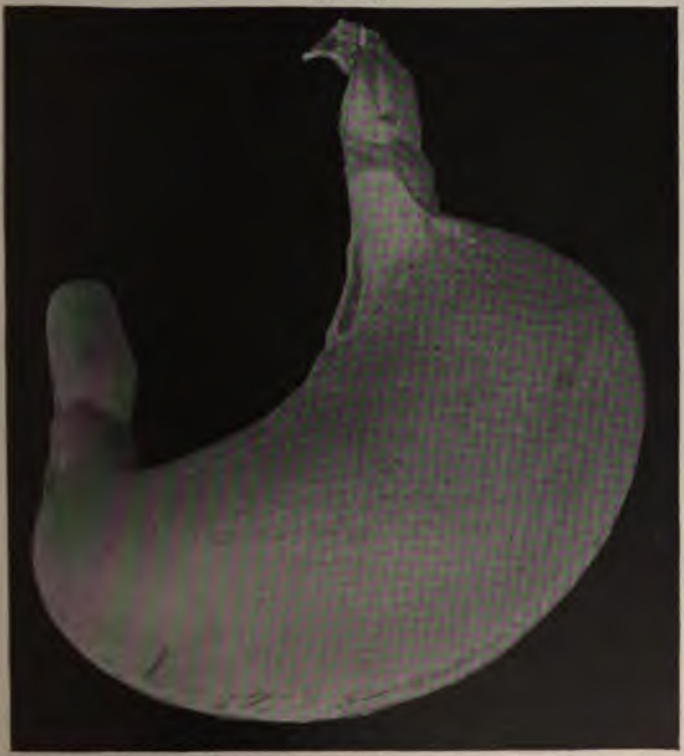
No rules for growth can be deduced from this very limited number of cases. The significance of these figures will be understood a little later when the feeding of the early days of life is discussed. Fig. 24 represents the stomach of an infant twelve days old. It represents the usual shape and position of the stomach in early life, and has been distended beyond

FIG. 23.



Stomach of infant 7 days old, natural size (posterior view). Gastric capacity, 40 c. c. Weight, 2700 grammes.

FIG. 24.

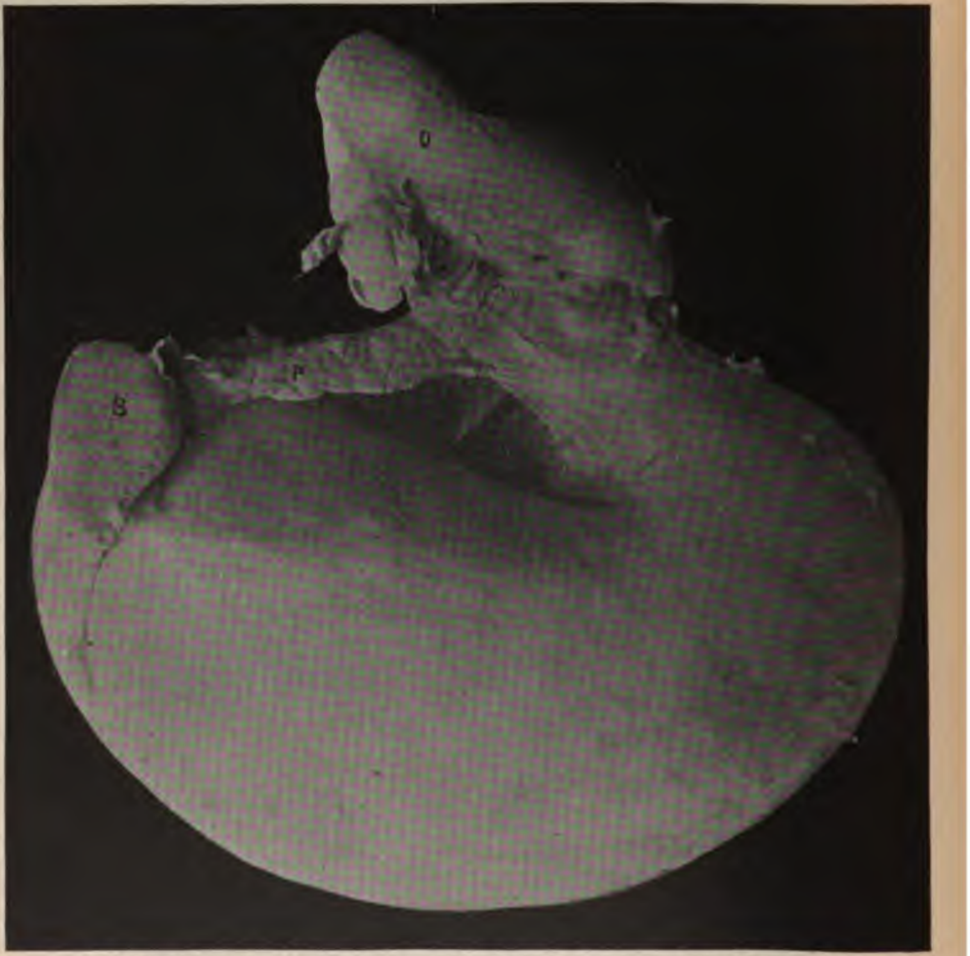


Stomach of infant 12 days old, distended to hold 80 c.c. Natural gastric capacity, 40 c.c.

the limit of its normal capacity to show the great elasticity of the ventricular walls already referred to. The gastric capacity was about 40 c.c. ($1\frac{1}{3}$ ounces); when distended it held 80 c.c. ($2\frac{2}{3}$ ounces).

Fig. 25 shows the stomach, duodenum, spleen, and pancreas of a well-developed infant five months old.

FIG. 25.

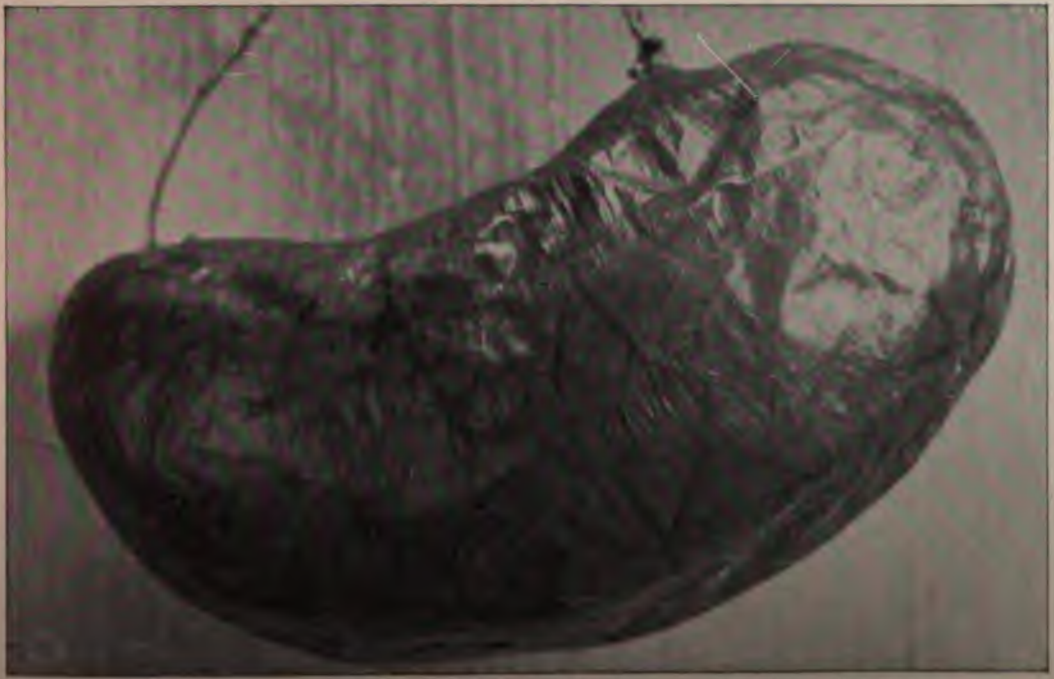


Stomach of infant 5 months old (posterior view). Weight, 6000 grammes. Distended to hold 225 c.c. Natural gastric capacity, 120 c.c. S marks the spleen; P the pancreas; D the duodenum.

Fig. 26 shows the stomach of an infant seven months old and weighing 5500 grammes (12 pounds). Its capacity is 220 c.c. ($7\frac{1}{3}$ ounces), which corresponds to its age rather than to the weight, which is that of an infant four months old.

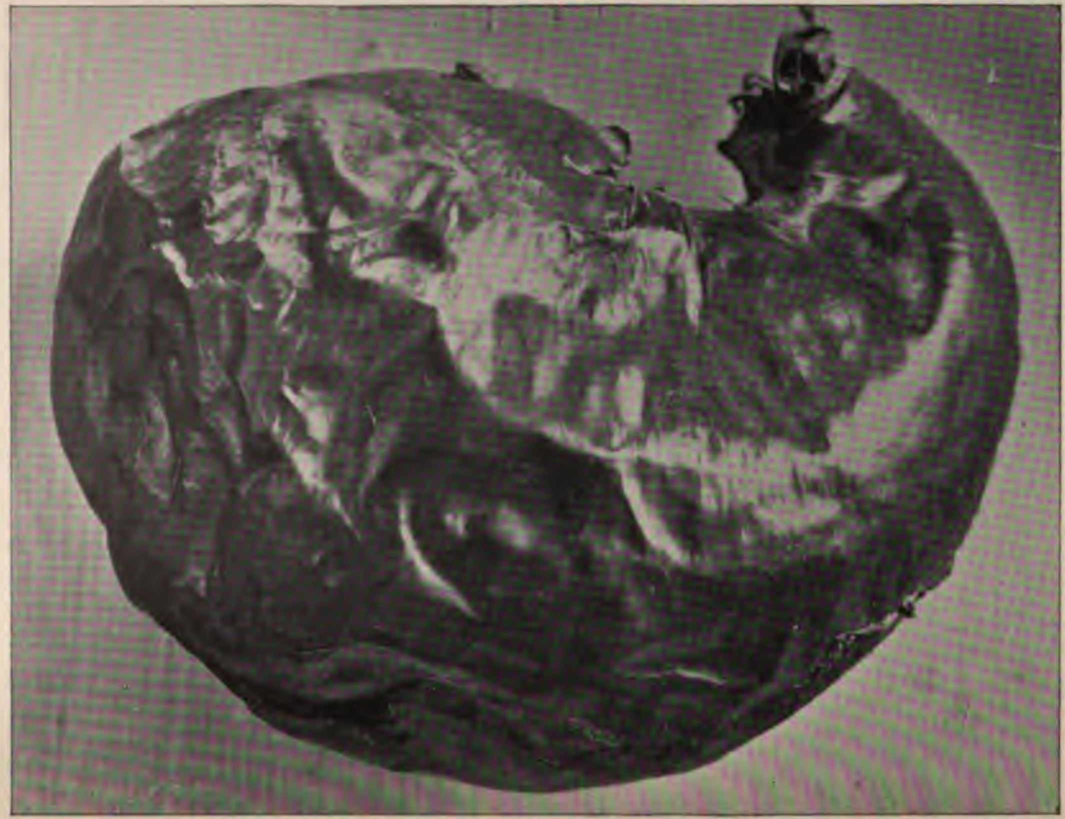
Another stomach taken from an infant also seven months old, but whose weight was that of an infant four months old, had a capacity of 150 c.c. (5 ounces), which corresponded to its weight rather than to its age.

Fig. 26.



Stomach of infant 7 months old, natural size. Weight, 5500 grammes. Gastric capacity, 220 c.c.
Dry preparation.

Fig. 27.



Stomach of infant 19 months old, natural size (posterior view). Weight, 6270 grammes. Gastric capacity, 300 c.c.

Fig. 27 shows the stomach of an infant nineteen months old and weighing 6270 grammes (13¾ pounds). Its capacity is about 300 c.c. (10 ounces).

According to these figures the gastric capacity in the third, fourth, and fifth months may appear rather small, and considerable differences will arise in the measurements by different observers. This, however, only emphasizes the fact that the problem of gastric capacity has not been solved by any system of measurement. When all observers have agreed to make use of a mathematically precise and constant pressure in measuring the stomach, we may possibly arrive at more uniform results. Even then the degree of elasticity will be found to differ so greatly in the individual stomach that most diverse measurements will result.

There is no doubt that the value of these calculations lies in making us recognize evident changes in the activity of growth at certain periods, in making us allow that great differences arise irrespective of age and weight, in impressing us with the fact that the gastric capacity has been over- rather than under-estimated, and in insisting that more exact clinical observations should be employed to reinforce our anatomical and physiological data.

Through the aid of a milk laboratory one may adapt exactly to the apparent needs of the infants, as well as to their age and weight, the amounts of food on which they have seemed to thrive.

The following figures represent the average amounts of food taken at different periods during their first year by three hundred and forty-one infants. They were all well and strong, of average weight, and all were thriving and steadily gaining during the year. They received only stated amounts of food carefully ordered by prescription at the milk laboratory, and were watched with the greatest care to see when they evidently were hungry enough to have the total amount of their food increased. Of course the opportunity for exact work is almost unlimited where one has a milk laboratory at his command, and it has therefore seemed to me that this method of determining the gastric capacity is an unusually good one, and one which has never thoroughly been carried out before. The following case explains the significance of the general figures:

An infant was fed with the greatest care both as to the quality and as to the quantity of its food. The following table represents the amount of food given at each meal from birth to ten months:

TABLE 11.

Amounts of Food in an Especial Case.

Age.	Cubic Centimetres.	Ounces.
Birth	30	1
4 weeks	45	1½
8 weeks	60	2
12 weeks	75	2½
16 weeks	90	3
20 weeks	132	4¼
6 months	150	5
7 months	150	5
8 months	150	5
9 months	195	6½
10 months	240	8

This case shows the necessity for frequent and great increase of the total amount in the first four or five months, the comparative quiescence of growth in the sixth, seventh, and eighth months, and the increase again in the ninth and tenth months. It does not, however, correspond so closely to my previous results as does this table, in which averages taken from the three hundred and forty-one cases already referred to are given.

TABLE 12.

Three Hundred and Forty-one Infants fed at the Milk Laboratory.

Age.	Number of Cases for each Age.	Average Amount of Food at each Feeding.	
		C.c.	Ounces.
Birth	45	29.4	0.98
4 weeks	76	70.5	2.35
8 weeks	84	96.6	3.22
12 weeks	97	118.8	3.96
16 weeks	87	137.0	4.57
20 weeks	86	158.4	5.28
6 months	73	171.3	5.71
7 months	56	185.4	6.18
8 months	54	208.5	6.95
9 months	45	226.2	7.54
10 months	33	238.8	7.89
11 months	28	242.0	8.07

In this table the same infant has of course been recorded a number of times at different ages.

The whole question of gastric capacity is so closely connected with the subject of infant feeding that it can be spoken of in detail later, when it will be seen to be of infinite importance in our attempts to regulate the substitute-feeding of infants.

INTESTINE.—**Small Intestine.**—During the first month after birth, it may be reckoned that the small intestine will grow about two feet (about sixty-one centimetres), and a like rate of growth may usually be recorded at the end of the second month of extra-uterine life; but after that period its development proceeds in a most irregular manner. Thus, in a child of one year the small intestine measured eighteen feet (about five hundred and forty-nine centimetres), while in another, aged two years, the length was only thirteen feet eight inches (four hundred and seventeen centimetres). Again, in a child aged six years the small intestine was no less than twenty-one feet (about six hundred and forty and five-tenths centimetres) in length, while in another child, eleven years of age, its length was fourteen feet (about four hundred and twenty-seven centimetres).

According to Treves, the great variations which appear so early in the length of the small intestine bear no relation to the growth of the child. They probably depend on the diet. Not only the quantity but the quality of the food is an important factor in the growth of the intestine. The amount of residue, also, and the more or less irritating qualities of the food, must all have their effect.

Peyer's patches are found very early. I have seen them at three days and again at thirteen days.

In another case, sixteen months old, Peyer's patches were found, and one of them was five inches long.

Large Intestine.—Treves has also observed that up to three or even four months the length remains the same, but that nevertheless a remarkable change occurs. This is that the large intestine grows at the expense of the sigmoid flexure, which at birth is nearly one-half of the large intestine, while at four months it has assumed about its permanent proportion. Treves found the large intestine to measure at one year two feet and six inches (about seventy-six centimetres); at six years about three feet (about ninety-one and five-tenths centimetres); and at thirteen years about three feet and six inches (about one hundred and seven centimetres).

Cæcum and Ascending Colon.—In about thirty-five observations on children under four years of age, most of them new-born infants, the cæcum was found in about thirty cases to range from the right lumbar region to the lower part of the iliac fossa. It was very frequently found at the junction of the rather vague lumbar and iliac regions. More or less would usually be found between two parallel horizontal lines, one at the level of the highest point of the crest of the ilium and the other at its anterior superior spinous process. In five cases the cæcum was either in the right iliac fossa or over the true pelvis, the fact being that it was so free as to have no fixed position. It is comparatively recently that the truth has been recognized in America, England, and France that normally the cæcum is at every age completely invested by the peritoneum, and that the idea that a large part of the posterior surface rests on areolar tissue without any intervening serous membrane is baseless, except in rare instances.

In young children the ascending colon differs in some respects from that of the adult. Owing to the high position of the cæcum, to say nothing of the relatively greater size of the liver, it is very short. There is no question that the ascending colon much more frequently has a mesentery than in the adult, and also that a relatively larger portion of the part above the cæcum is also invested with peritoneum so as to be absolutely free. Dwight believes that the cæcum of the infant and that of the young child are much more movable than that of the adult, and are also usually situated higher.

Vermiform Appendix.—The length and direction of the vermiform appendix are very variable. I have found it six and a half centimetres (two and five-eighths inches) long in a girl of thirteen days, five and three-tenths centimetres (two and one-eighth inches) in one of three years, eight centimetres (three and one-quarter inches) in one of ten months, and seven and a half centimetres (three inches) in a girl eleven weeks old. It would appear from Treves's researches that the fœtal shape of the cæcum is that of a pouch hanging down from the point of junction of the small and the large intestine and continued into the appendix, which grows symmetrically from the middle. Later, however, an irregular growth of one side of the cæcum generally leaves the origin of the appendix near the end of the ileum. Dwight has found that this condition usually occurs in the child. The position and direction of the appendix are most uncertain. It is, however, as a rule, on the posterior side of the cæcum. Its little mesentery passes to its beginning from the cæcum and is only exceptionally attached to the walls of the abdomen or pelvis.

The importance of the lymphatic glands about the cæcum as possible starting-points of inflammation is very great. Tuffier states that the lymphatics of the front of the cæcum follow the anterior ileocæcal artery to empty into two glands which he has found constantly in the superior ileocæcal fold, and which are very distinct in the child. The posterior glands are also found constantly on the posterior and inner wall of the cæcum itself beneath the peritoneum. They usually form a group of from three to six.

Sigmoid Flexure.—In some cases, according to Dwight, the sigmoid flexure is obviously very long, in others apparently of about the adult relative proportions. Even in infants, in whom the sigmoid flexure does not, as a rule, seem large, it often has a relatively broad mesentery, allowing free displacement.

Descending Colon.—As is well known, the descending colon usually has no mesentery, but still one is often found. Lesshaft, in his observations made on subjects of many different ages, found it once in six times. Dwight, in rather more than twenty infants, found a mesentery to the descending colon in about half the cases. Lesshaft found a mesentery less often in young subjects than in others. A great part of the large intestines in infants is less fixed than in adults.

TEMPERATURE.—The temperature of the infant at term, although varying within a slight limit, is usually slightly raised. Very soon, however, as would be expected from the tax which is immediately made on its vitality by so many new surroundings, the temperature falls rather below the normal adult standard. In about a week the normal infant has recovered its equilibrium, and, if its nutriment has also been properly adapted to its digestive power, it usually presents the average normal adult temperature, 36.8° C. (98.2° F.).

TABLE 13.

Temperature of Infant at Term.

At birth	37.2° C. (99° F.).
Within an hour	36.1°–35.5° C. (97°–96° F.).
In about a week	36.8° C. (98.2° F.).

These figures are the average of a large number, and are subject to great variations, as is seen on comparing them with a number of observations undertaken at my request by Dr. C. W. Townsend at the Boston Lying-in Hospital:

TABLE 14.

Townsend's Temperature Observations.

Age.	Temperature.	Age.	Temperature.
1 day	37.2° C. (99° F.).	9 days	37.4° C. (99.4° F.).
2 days	37.3° C. (99.2° F.).	9 days	37.1° C. (98.8° F.).
5 days	36.6° C. (98° F.).	9 days	36.9° C. (98.4° F.).
5 days	37.5° C. (99.5° F.).	10 days	37.1° C. (98.8° F.).
6 days	37.3° C. (99.1° F.).	13 days	37.2° C. (99° F.).
7 days	37.5° C. (99.5° F.).	13 days	37.3° C. (99.2° F.).
7 days	37.2° C. (99° F.).	16 days	37.3° C. (99.2° F.).
7 days	37° C. (98.5° F.).	20 days	37.3° C. (99.2° F.).

PULSE.—The pulse in uterine life is, as a rule, somewhat higher in girls than in boys, the former being about 130 to 140, and the latter 120 to 130. Anything over 130 points towards the female sex, but these figures as a means of distinguishing the sexes before birth are not to be relied upon. At birth the pulse soon falls somewhat, and may be quite irregular. This, as a rule, is merely what we should expect would be the result of the sudden and great change which has taken place in the circulatory mechanism, and of the additional force which the heart is called upon to supply when it becomes the central station from which the blood is propelled. The lungs also are scarcely ready to perform at once their function, and are often somewhat more of an obstruction than an aid to the blood-current. The pulse in early life, especially during the first year, varies very much, but, as a rule, allowing that the girl's pulse is usually more rapid than the boy's, the following table represents pretty well what may be expected in males.

TABLE 15.

Pulse-Rate for Males.

Age.	Pulse-Beats per Minute.
Early weeks	120 to 140
Until 2d year	110
2 to 3 years	100
5 to 8 years	90

From the eighth year up to puberty the pulse gradually acquires the adult rate. The pulse in children varies greatly under the many nervous influences which are continually affecting it in early life.

Townsend has also made a record of the pulses taken in the same infants whose temperatures were recorded in Table 14. They do not especially correspond with the general averages, but are what may be expected in cases seen at random.

TABLE 16.

Townsend's Pulse Observations.

Age.	Quiet.	Crying.
1 day	130	158
2 days	120	156
5 days	152	164
5 days		160
6 days		152
7 days	120	154
7 days		160
7 days		152
9 days	148	
9 days	160	180
9 days	156	
10 days	152	
13 days	136	
13 days		168
16 days	168	172
20 days		168

RESPIRATION.—The respiration, although quicker in early life than in adults and corresponding somewhat to the pulse, assumes the equilibrium of a later period of development much earlier than is found to be the case with the pulse. It varies with changes of temperature and with excitement, and has its rhythm much more easily affected by diseased conditions than in later life. The following table represents fairly well what is usually found on counting the respirations when a child is quiet:

TABLE 17.

Respirations.

Age.	Respirations per Minute.
At birth	45
Until the 3d year	15 to 40
3 to 5 years	25

The following is the record made from observations on a healthy male eight months old, when he was lying quietly on his nurse's lap:

The type of respiration was decidedly abdominal. Counting the respirations by the rise and fall of the ensiform cartilage, which stood out quite distinctly, the respirations varied from 50 to 70 in the minute. They were, also, quite irregular, and by making with a pencil an upward stroke for every inspiration, a downward stroke for every expiration, and a horizontal line for every pause, the same lack of rhythm was found that appeared in the infant at term, described on page 36, and also the rhythm corresponding to that of the infant nine months old which is described on page 53.

Townsend has also observed for me the respiration of four healthy infants at the Lying-in Hospital, with the following results:

TABLE 18.

1.	Age, 1 hour	Respirations,	48 to 56.	(Awake.)
2.	Age, 2 days	"	30 to 52.	(Asleep.)
3.	Age, 3 days	"	24, 32, 44.	(Asleep.)
4.	Age, 6 days	"	28 to 40.	(Crying.)

The respiration in all these cases was very irregular, and both abdominal and thoracic in type. In the baby two days old the respiration was chiefly abdominal.

HEIGHT.—The average height of the male infant at term, is, according to a large number of measurements made by Quetelet, Vierordt, and others, about 49.5 cm. (19¾ inches). These figures correspond very closely to those which I have myself measured. Insufficient nourishment and improper food, especially as represented in rhachitic children, seem to retard the growth, while on the contrary, the various fevers seem to increase the activity of growth in length, while decreasing the total weight. In the first three or four months the growth is proportionally rapid to that in the latter part of the first year. In like manner the activity is greater in the first month than in the second, and in the second than in the third, becoming still less in the fourth, fifth, and six months.

The average increase for the first month is about 4.5 cm. (1¾ in.).
" " " " " second month is about 3.0 cm. (1½ in.).
" " " " " third to the fifteenth month is about 1 to 1.5 cm. (½ to ¾ in.).
" " " " " first year is about 20 cm. (8 in.).
" " " " " second year is about 9 cm. (3½ in.).
" " " " " third year is about 7.4 cm. (3 in.).
" " " " " fourth and fifth years is about 6.4 cm. (2⅝ in.).
" " " " " fifth to the sixth year is about 6 cm. (2⅜ in.)

The height is about doubled in the first six years, and at fourteen years the final height has usually been attained to within about one-twelfth. The height at different ages will be shown in comparison with the weight on page 81. The growth in height seems to be most active in the spring.

WEIGHT.—In quite a number of cases it has been found that the careful and systematic weighing of infants gives warning of the approach of disease some days before any other symptoms are evident. This point was very clearly illustrated in a case which was under my care at the Infants' Hospital. The infant entered the hospital to have its food regulated. It was apparently perfectly well, but after a few days the daily weighing showed that it was losing. The loss of weight continued to be the only perceptible symptom for a number of days, when it manifested certain nervous phenomena and died a few days later of *cerebral thrombosis*. We sometimes notice a loss in weight preceding a chronic nutritive disturbance by several weeks, and if the coming disease is an acute one, or is of unusual severity, the loss is often sudden and great. The careful and systematic weighing of children may be of considerable value, therefore, in preventive medicine. Thus, if we have noticed that a child has without perceptible cause lost weight, we can, by guarding it from an exposure which in health would not be too great, prevent it from having complications such as of digestion or from cold, and render the coming disease milder in its type and more readily dealt with. In a paper on the Relation between Growth and Disease, by H. P. Bowditch, these changes in weight are especially dwelt upon, and it is apparently shown that this method of determining the onset of the disease is more useful in chronic than in acute diseases, though even in the latter class it is not impossible that the warning may be given in time to be of use, and to merit the term of "danger signal" which has been given to it by Bolton. Bowditch shows in the following table the rate of growth of a girl between two and three years old, and the relation between growth and disease. The figures represent the absolute weight of the child obtained by weighing in the ordinary manner, and then deducting the weight of the clothes:

TABLE 19.

DATE.	Age, in Weeks.	WEIGHT.		
		Kilo.	Lbs.	
1880.				
September 19	107	11.40	25.08	
October 3	109	11.40	25.08	
November 7	114	11.78	25.91	
December 5	118	12.25	26.95	
December 12	119	12.28	27.01	
December 26	121	11.90	26.18	
1881.				
January 2	122	12.15	26.73	
January 23	125	11.80	25.96	
January 30	126	11.65	25.63	
February 6	127	11.55	25.41	Enlarged cervical glands noticed February 5.
February 13	128	11.55	25.41	Clay-colored dejections February 12–15.
February 20	129	11.95	26.29	
February 27	130	11.75	25.85	
March 6	131	11.94	26.26	
March 13	132	12.15	26.73	
March 20	133	12.20	26.84	
March 27	134	12.41	27.30	
April 3	135	11.91	26.20	Attack of measles beginning April 5.
April 10	136	11.71	25.76	
April 17	137	11.98	26.35	
April 24	138	12.00	26.40	
May 1	139	12.03	26.47	
May 8	140	12.01	26.42	
May 15	141	12.34	27.14	
May 22	142	12.15	26.73	Cold in the head beginning about May 22.
May 29	143	12.09	26.60	

An examination of this table shows that the child, having grown rapidly during the autumn, suddenly, and without any manifest cause, began to lose weight about the middle of December. This loss of weight was irregularly progressive until February 6, when an enlargement of the cervical lymphatic glands was noted, followed a week later by clay-colored dejections. These symptoms yielded to appropriate treatment, and the child again gained weight rapidly until March 27, when a sudden loss of weight occurred, followed by an attack of measles. A subsequent loss of weight in May seems to have been associated with a rather severe cold in the head. We have here, then, a case in which a disorder of nutrition manifested itself by enlarged glands and by clay-colored discharges, but in which these symptoms were preceded for several weeks by a progressive loss of weight. It seems not unreasonable to suppose that this loss of weight was the first symptom of a disturbance which afterwards manifested itself by more unequivocal signs. Even in the case of the acute attack of measles it will be noticed that the loss of weight preceded by at least a week the actual eruption of the disease. It must not be supposed, however, that loss of weight in a growing child is in every instance a precursor of actual disease. The weight of a healthy child is liable to oscillation within limits which have not been accurately determined, but it may sometimes amount to ten or fifteen per cent. in a week. Children lose in weight and regain their loss in a wonderful manner, so easily are they affected by even slight physical disturbances, and so great are their recuperative powers. The weight of boys, as a rule, is somewhat greater than that of girls at birth, and remains greater up to the age of puberty, when the girl rapidly overtakes the boy, surpasses him, and becomes a developed woman very soon, while the boy does not become a man until some years after puberty. This fact is exemplified in the table on page 81, which shows that the girls have surpassed the boys in their height at the eleventh year, and in their weight at the twelfth year, when they are found to be taller and heavier than the boys, as is the case also in the thirteenth and the fourteenth year.

The systematic and frequent weighing of infants during the first year of their lives I consider to be of great importance, and far more useful as a means for determining their nutritive condition than any other one method of which we know. For many years I have had the infants at the Infants' Hospital weighed every day as regularly as they are fed, and a glance at the column containing their weights in the various weeks and months gives information as to their general health, and serves as a guide to the changes which it may be necessary to make in their food. The information gained in this way is far beyond what the most careful physical examination could disclose. The weight is, in fact, an index of the nutritive processes to such an extent that it is representative of the child's well-being, while the height gives us information rather as to its cellular activity. The normal average weight of quite a number of infants at term is for males 3250 grammes (7½ pounds), and for females 3150 grammes (7 pounds); many individual cases occur, however, where the weight is either greater or less than these figures, and yet the infant is healthy. The increase in weight is in direct proportion to the original weight, and if the original weight is small the gain is usually correspondingly small. This, however, is only a general rule, for at times infants of light weight are met with whose gains are remarkably large, and often surpass those of infants with a heavier initial birth weight. During the first three or four days of life there is usually a loss in weight, and the original weight is in a large number of cases regained only in the second week. If it is not regained by the third week, we should consider that it is a warning that the nutrition of the infant is at fault, and that especial measures should be taken to increase its vitality. This initial loss of weight is usually designated as *physiological*. We must, not, however, be misled by this term, or place too much confidence in it, for, as a rule, this initial loss, which often amounts to from 270 to 300 grammes (9 to 10 ounces) can be accounted for only partially by natural physiological causes. The additional loss is evidently pathological, and is to be so regarded, in order that we should endeavor to obviate it, and thus prevent imposing an additional tax on the infant's vitality at a time when any tax whatever should be regarded as serious. Townsend has made some interesting investigations on this loss of weight at the Boston Lying-in Hospital, which show that the infants of primiparæ lose about 45 grammes (1½ ounces) more than those of multiparæ; also, deducting 45 grammes (1½ ounces) as the average loss from removal of the vernix caseosa, the meconium still remaining, that the loss in weight is reduced to 247 grammes (8⅕ ounces) in the infants of primiparæ, and to 222 grammes (7⅗ ounces) in those of multiparæ. The whole loss should include the meconium, which is computed to weigh about 60 to 70 grammes (2 to 2⅓ ounces), so that a loss of from 90 to 150 grammes (3 to 5 ounces), which includes also the urine, on the first day, can, in a very general way, be admitted to be purely physiological. Townsend's figures also show that although the infants of primiparæ lose more and are slower to recover the loss than are those of multiparæ, yet after the second week they overtake and keep pace with the latter. The whole question is simply one of nutrition, it being well known that the milk of primiparæ is somewhat longer in acquiring its equilibrium than that of multiparæ, but that finally it is equally nutritious. It was also found that the presence of the colostrum corpuscles in the milk had something to do with the loss or with the failure to gain. Where the colostrum persisted the infants lost more than when it speedily disappeared. The colostrum should normally disappear in the first week. Where its presence is prolonged into the third week, the infants do not thrive. Three cases at the hospital illustrated this point: all the mothers seemed healthy and had plenty of milk.

(1) Multipara—no colostrum on third day,—infant's loss 8 ounces.
(2) Multipara—colostrum until ninth day,—infant's loss 16 ounces.
(3) Primipara—colostrum until thirteenth day,—infant's loss 14 ounces.

The average loss in five infants of multiparæ in whose milk the colostrum was absent by the fifth or sixth day was 10 ounces.

The whole nervous system of the young child is much more active and excitable than that of the adult. The brain, for instance, besides being fifteen times as large proportionately in the infant as in the adult, increases with much greater rapidity up to the age of seven years than at any other period. In connection, probably, with the constructive labors of the growing tissues is the activity of the lymphatic system. The absorption of oxygen is said to be relatively more rapid than the production of carbonic acid,—that is, there is a continued accumulation of capital in the form of oxygen-holding compounds. The food represents so much potential energy, but it must be converted into tissue before the energy can become vital, and in such conversion a large amount of molecular energy must be expended. The metabolic activity is more pronounced in the infant than in the adult, and is expended not so much on the energy required in the external world as for the rapidly increasing mass of tissue. Another reason for the presence of more active metabolism in the infant than in the adult is the necessity of rapid molecular interchange to keep up the temperature. The infant having the smaller body, and yet the relatively larger surface (the extent of skin thus being proportionately greater), it loses more heat proportionately than does the adult, and thus suffers more easily from changes of temperature (Foster).

Disturbances of the nutritive process from these conditions very easily arise, and the process of assimilation is much more important than in adult life, for the child's activity implies a greater consumption of nutriment in the form of food or tissue. The child's equilibrium is thus much more easily disturbed than the adult's, and this creates a greater susceptibility to disease and less power to resist external influences. This is well exemplified by the rule that the younger the individual the greater the mortality. There are three times as many deaths in the first half of the first year as in the second half, and a large proportion of those dying in the first half-year die in the first month. Of those dying in the first month, death occurs in a large proportion in the first week. A considerable number of the deaths which occur in the early weeks of life, especially in the first week, are from asthenia. These facts are very significant in connection with the child's loss of weight in the early days of life over that which we have just described as being physiological. Lack of sufficient nourishment and an unstable equilibrium are the factors in the problem which represent this early loss of weight. These conditions are enhanced by the state of the mother, who, exhausted by the process of labor, is not able to supply a food for her infant which is adapted to its sensitive and incompletely developed digestive function. (Evetsky).

In addition to these manifest causes for loss of weight, we must consider that the new-born infant is much more susceptible to external impressions than when after the first weeks its various functions have become adapted to their new surroundings.

The whole system is stimulated to greater activity of tissue interchange not only by the sudden change of temperature to which the skin is exposed, but also by the change from darkness to light, and from silence to a greater or less degree of sound. This transient early period of life, therefore, is marked by a superactive metabolism, insufficient nourishment, and resulting asthenic conditions which are analogous to starvation. This is represented as a whole by a loss of weight evidently of a pathological character, in addition to that which has been described as physiological. One will, therefore, understand with what care the newly born infant should be protected from too great changes of temperature, too much light, and too much noise. The analogy of this statement is found in the sensitive organization and habits of the lower animals. In this way only can the digestive function be made to correspond to such an extent, in the early days of life, to the work which is required of it, as to keep the loss of weight within the physiological limit. Starvation, as is well known, proves fatal primarily not from the amount of food furnished being too little for the processes of disintegration, but from exhaustion of the nervous system. The endurance of the starvation is in proportion to the capability of resistance of the nervous tissue. This nervous tissue is so highly sensitive and has such great functional activity in the infant, proportionately to the adult, that it needs much more nourishment, and succumbs much more quickly to deprivation from nourishment. Young animals die in a very much shorter time when deprived of food than do older ones from this cause. It is not surprising, therefore, that when the early period of life is represented only by hours and days, the various disturbances which would be of minor consequence at a later period of existence should have a decidedly pathological effect and produce a marked loss in weight beyond the natural physiological loss. The following case exemplifies the practical bearing of what has just been said.

A male infant was born December 16 at term. It was healthy and vigorous, and gave no evidence of organic disease. The mother, a multipara, strong and healthy, was twenty-eight years of age. Her other children were living and healthy. On the third day, December 19, the infant had a slight attack of icterus neonatorum, which disappeared in twenty-four hours. On the fifth day, December 21, the weather was very cold and bleak, but the infant was taken to church and christened. The church was warm and the infant reasonably well protected from cold, but there was a large number of people present, and an unusual amount of noise. The infant, on being taken home, immediately began to show symptoms of asthenia, and on the following day was found to be cyanotic and breathing rapidly, with a subnormal temperature and no apparent organic disease. It died in the afternoon. The asthenia seemed to be produced by too early exposure to change of temperature, light, and sound.

As a rule, the average daily gain in the first two months should not be below twenty grammes (two-thirds of an ounce). It has been found at the Infants' Hospital that if the gain is less than this the infant, as a rule, is being badly nourished, is sick, or is going to be sick. There are, of course, exceptions to this rule, and it should be clearly borne in mind that observations of weight including only that of two or three days are very misleading, and that it is the week's weight which gives us the fairest idea of loss or gain. Thus, one frequently finds infants showing a daily gain of only five or ten grammes (one-sixth or one-third ounce), or even losing fifteen or thirty grammes (one-half or one ounce) on one day, and then gaining one hundred to one hundred and fifty grammes (three and one-third to five ounces) on the next day. From this it will be readily understood that we should obtain from one day's observation too low and on the next day too high an estimate of the nutrition. By the end of the week, however, the weights usually equalize each other, and we have fairly correct figures to guide us. The following table shows about what would be expected of the average infant as to weight during the first year. Girls, as a rule, gain less than boys, but this is only if they are of lighter initial weight. The heavy girls make the same large gains as the heavy boys, but, as a rule, their initial weight is smaller than that of the boys, and they therefore make smaller gains.

TABLE 20.

General Figures of Weight.

Age.	Weight.		Average gain per day.	
	Grammes.	Pounds.	Grammes.	Ounces.
At birth	3000 to 4000	6.6 to 8.8		
From birth to 5 months			20 to 30	⅔ to 1
From 5 months to 12 months			10 to 20	⅓ to ⅔

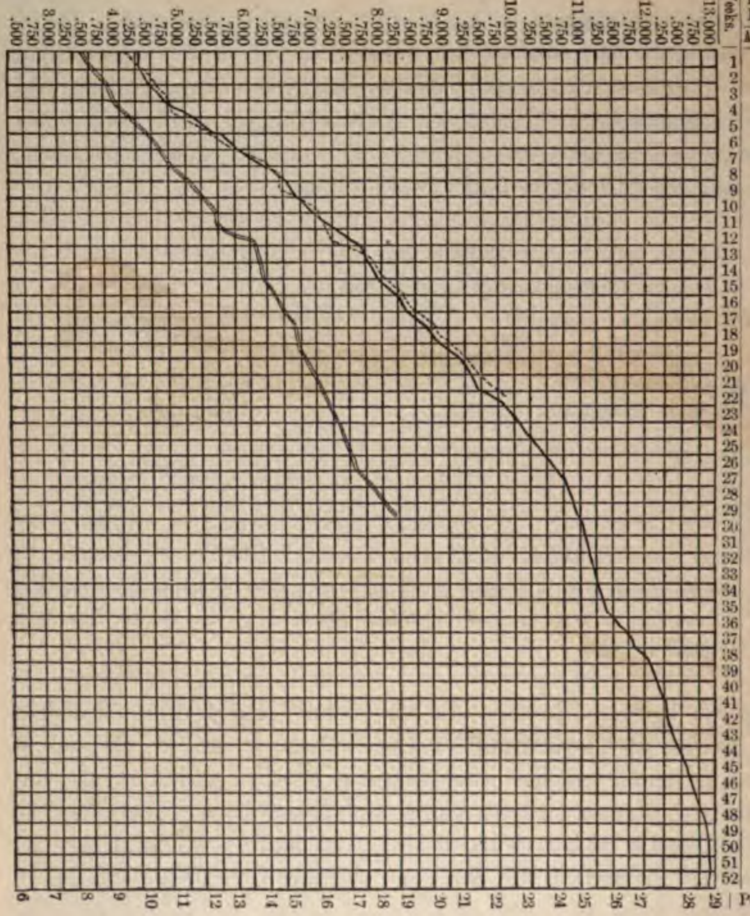
Age.	Weight.	
	Grammes.	Pounds.
At 1 year	9,500	20.90
At 7 years	19,000	41.80
At 14 years	38,000	83.60

[The above figures are on a basis of 3500 grammes (7.7 pounds) at birth, and of a gain of 30 grammes per day for the first four months and 10 grammes per day for the last eight months of the first year.]

Useful figures to remember are that the initial weight is doubled at five months and trebled at fifteen months; also that the weight at one year is doubled at seven years, and that this weight is again doubled at fourteen years. There are, of course, both gains and losses in weight during the year, the weight acting as an index of the disturbances which arise. As a rule, what may be called the line of nutrition rises from the initial weight in the first week up to the fifty-second week. A uniform increase is, however, exceptional, on account of the many disturbances, such as from food, the dental periods, weaning, improper hygienic care, and diseases.

Instances of continual weekly gains during the first year have occasionally come under my notice in both hospital and private practice, and the chart on page 80 gives the exact weights of a healthy male infant fed by a wet-nurse for over a year, and will serve as an example of the ideal line of nutrition. The infant was gaining so regularly that the weighing was omitted for several weeks, which was unfortunate, as the weights would probably have shown the same uniform gain. A weekly gain is also shown in this same chart of a male and a female infant, brother and sister, nursed by their mother. The double line represents the boy's weights in the first twenty-nine weeks of his life; and the dotted line the girl's weight for twenty-one weeks.

CHART 1.



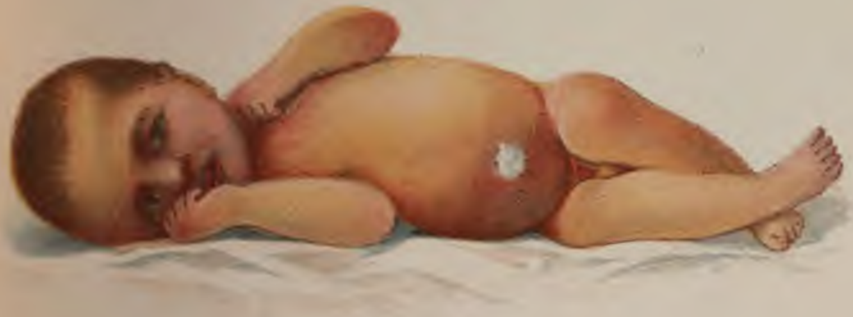
Name, ...
Date of Birth, June 21.
Initial Weight, 4650.

Weeks.	Actual Wt.	Date of W'g.
1	4,500	June 27.
2	4,612	July 4.
3	4,916	" 11.
4	5,332	" 18.
5	5,684	" 25.
6	6,004	Aug. 1.
7	6,292	" 8.
8	6,644	" 15.
9	6,852	" 22.
10	7,172	" 29.
11	7.476	Sept. 5.
12	7,802	" 12.
13	7,994	" 19.
14	8,170	" 26.
15	8,362	Oct. 3.
16	8,586	" 10.
17	8,912	" 17.
18	9,136	" 24.
19	9,376	" 31.
20		
21	9,968	Nov. 14.
22	10,912	" 21.
23		
24		
25		
26	10.912	Dec. 20.
27		
28		
29		
30		
31		
32		
33		
34		
35	11,680	Feb. 20.
36	11,904	" 27.
37	12,032	March 5
38		
39		
40		
41		
42	12,544	April 9.
43		
44		
45	12,640	April 30
46		
47		
48		
49		
50		
51	13,104	June 1.
52	13,376	" 20.

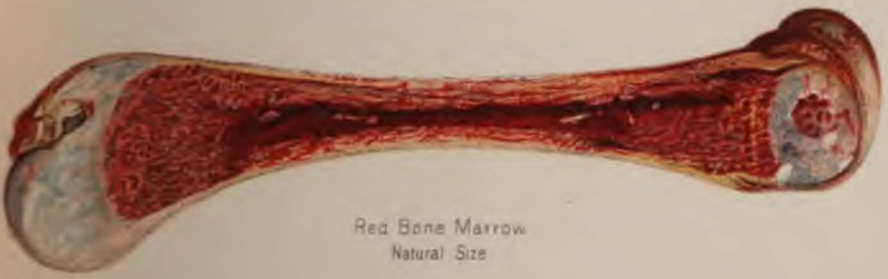
The figures for birth, for five months, and for one year represent my investigations, combined with the figures which have already been shown. The figures for the second and third years are taken from a series of investigations made by Peckham, of Milwaukee, in the Report of the Wisconsin State Board of Health for 1882. The figures for the fourth year are approximate averages taken from children of three and five years. The figures from the fifth year to the fourteenth year were taken from Bowditch's article on the Growth of Children, in the Twenty-second Annual Report of the State Board of Health of Massachusetts. They represent the average figures of a large number of school-children.

In the following table the weights at birth, and in the first, second,

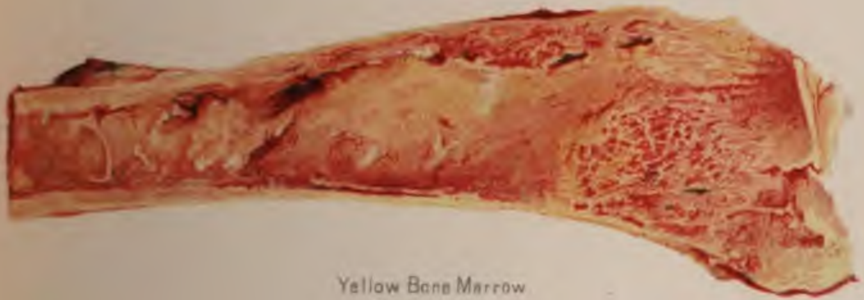
PLATE II



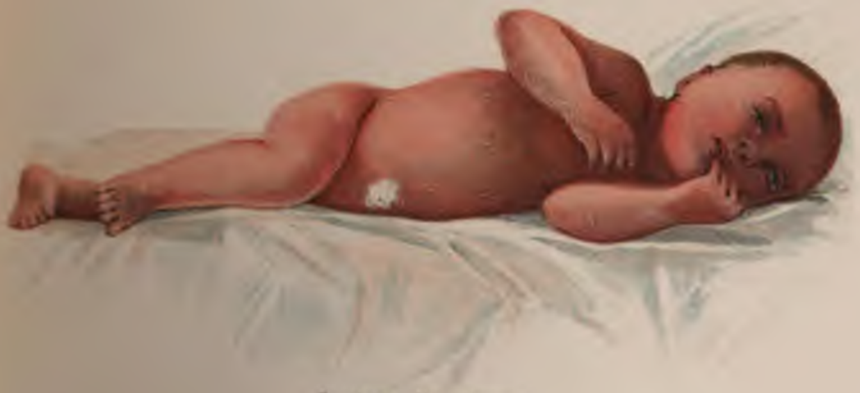
Icterus Neonatorum.



Red Bone Marrow
Natural Size



Yellow Bone Marrow
¾ Natural Size



Erythema Neonatorum.

and third years, were taken without clothing. The ordinary school-clothes were worn in the weighing from five to fourteen years.

TABLE 21.

Average Heights and Weights from Birth to Five Years, and of Boston School Boys and Girls, irrespective of Nationality, from Five to Fourteen Years.

Boys.				Age.	Girls.			
Height.		Weight.			Height.		Weight.	
Centimetres.	Inches.	Kilogrammes.	Pounds.		Centimetres.	Inches.	Kilogrammes.	Pounds.
49.87	19.75	3.25	7.15	Birth.	48.12	19.25	3 15	6.93
61.87	24.75	6.50	14.30	5 months.	59.12	23.25	6.30	13.86
73.82	29.53	9.54	20.98	1 year.	74.17	29.67	9.00	19.80
84.55	33.82	13.80	30.36	2 years.	82.35	32.94	13.31	29.28
92.65	37.06	15.90	34.98	3 years.	90.77	36.31	15.07	33.15
98.27	39.31	17.27	37.99	4 years.	97.00	38.80	16.53	36.36
103.92	41.57	18.64	41.00	5 years.	103.22	41.29	17.99	39.57
109.37	43.75	20.49	45.07	6 years.	108.37	43.35	19.63	43.18
114.35	45.74	22.26	48.97	7 years.	113.80	45.52	21.50	47.30
119.40	47.76	24.46	53.81	8 years.	118.95	47.58	23.44	51.56
124.22	49.69	26.87	59.00	9 years.	123.42	49.37	25.91	57.00
129.20	51.68	29.62	65.16	10 years.	128.35	51.34	28.29	62.23
133.32	53.33	31.84	70.04	11 years.	133.55	53.42	31.23	68.70
137.77	55.11	34.89	76.75	12 years.	139.70	55.88	35.53	78.16
143.02	57.21	38.49	84.67	13 years.	145.40	58.16	40.21	88.46
149.70	59.88	42.95	94.49	14 years.	149.85	59.94	44.65	98.23

FEET.—According to Dane, the pad of fat that fills the hollow under the bones in the infant's foot is designed to support the arch until the muscles are strong enough to hold it by themselves. The fat in this way acts as a kind of physiological flat-foot plate.

As soon as the muscles become strong enough to protect the arch the pad of fat is no longer necessary, and is slowly absorbed, until by the fourth or fifth year the foot presents the same appearances found in the adult.

In a few children the pad of fat is found to be wanting, and in many of these cases the arch is so far obliterated that the tubercle of the scaphoid may even rest on the ground. These latter are the true cases of "flat-foot."

BONE MARROW.—The change from the red bone marrow of early life to the yellow marrow of a later period begins, according to Minot, before birth, and progresses in each bone from the centre towards the periphery, or in the long bones towards the ends.

It begins earlier in the distal bones and then goes on from bone to bone centripetally. Concerning the exact time when these changes take place very little is known, and nothing definite. Plate II., facing page 80, shows the red and yellow bone marrow.

SKIN.—As the infant grows older the skin changes gradually to a delicate pinkish white in place of the decided pink of the early days of life.

CORD.—The cord should be carefully wrapped in antiseptic absorbent cotton, and no water should be allowed to come in contact with it. It will thus become dry sooner, and will gradually loosen and fall off.

FUNCTIONS.—The different functions of the infant, like its physical characteristics, vary considerably as to the time of their development. It is difficult, therefore, to give exact average figures, and, in fact, my observation of individual cases has differed so often from these average figures that we must allow much latitude in stating the proper time for an especial function to develop.

VOICE.—During the first year of its life the average infant uses its voice merely in crying to express its discomforts and desires. At about the twelfth month it usually begins to enunciate single words, and in the middle or towards the end of the second year it learns to form short sentences. Children vary very markedly as to the time when they really learn to talk connectedly, but this is usually accomplished by the third or fourth year, though it is somewhat later before they master the details of language, such as the proper use of prepositions.

MENTAL IMPRESSIONS.—The infant seldom SMILES before the fifth or sixth week, the change of expression of the mouth before that time being usually an indication of some discomfort. In individual cases, however, there is no doubt that the true smile of enjoyment comes earlier, even by the fourth week. The infant usually does not RECOGNIZE objects before the sixth or eighth week. Its HEARING is soon established. The functions of TOUCH, TASTE, and SMELL are apparently more or less developed at birth.

LACHRYMAL GLANDS.—The development of the function of the lachrymal glands varies considerably, but the infant will usually be found to shed tears when it is three or four months old. I have known tears to appear as early as the first month. They do not at first come every time the infant cries, so that a number of observations must be made on the same individual before deciding whether this function is present. I have also noticed that even older infants do not shed tears with each crying-spell. These facts are at times quite important to remember, as a suppression of the lachrymal secretion occurs where the infant's vitality has been profoundly affected by disease, and a return of the tears is an indication for giving a favorable prognosis, and often that convalescence is about to be established.

SWEAT GLANDS.—The sweat glands are developed at about the third to the fifth week. I have seen an infant in the second week of its life suffering so much as to have its circulation seriously interfered with from the high temperature of a bath-room where it was being bathed, while the nurse who was bathing it was perspiring profusely, and was apparently perfectly comfortable. There is, however, a great variation in the time when these glands develop, and at times even in the second week of life I have noticed cases where the head was seen to perspire quite freely. In certain individuals, therefore, the function of the sweat glands is fairly developed at birth.

Salivary Glands.—The saliva is a secretion which is somewhat slow in being established, both in quantity and in its amylolytic property, There is not much flow of saliva in the infant's mouth for the first three or four months of its life, and even when the function of the glands has become so developed that the saliva appears in the mouth in abundance, a comparatively small amount reaches the stomach by being swallowed. It flows out of the mouth over the chin, and until the latter part of the first year, when its amylolytic action has become established, it probably plays but an insignificant rôle in digestion. The salivary secretion contains a certain amount of ptyalin, but its diastatic powers seem to be in process of development, and this should indicate to us that this function ought not to be forced into use in digestion until it has become much better established, as towards the end of the first year.

Pancreas.—The amylolytic action of the pancreatic secretion is but little, if at all, developed at birth. Towards the end of the first year the function seems to have become fairly well established, and to a degree which will not be harmed by a moderate call upon it for the digestion of small quantities of starch. The pancreatic power of digesting fat also seems to be slight in the early months of life, but to increase gradually and to be well established by the end of the first year.

Bile.—The large size of the liver at birth and during infancy is well adapted to the great metabolic activity which is needed for the development of this period of life. The investigations of Jacubowitsch and Baginski show that the bile in children is poor in inorganic salts, with the exception of the iron salts. It is also distinguished by its small amount of cholesterin, lecithin, and fat, and the smaller percentage of its glycocholic and taurocholic acids, as compared with the bile of later life. The bile acids have the property of arresting pepsin digestion, and thus the small amount of bile acids present in infancy favor the action of the gastric juice, whose feeble ferment would otherwise be neutralized. The incomplete assimilation in infancy of very fatty food, such as creamy milk, is due to this lack of bile acids, for the latter aids in the emulsification of fat and its decomposition into fatty acids and glycerine. The feeble antiseptic properties of the bile in infancy and the resulting danger of intense fermentation of the intestinal contents is due to the lack of bile acids.

LYMPHATIC SYSTEM.—The high development of the lymphatic system in early life is very marked. According to Foster, not only are the lymphatic glands largely developed and more active than in the adult (as is probably shown by their tendency to disease in youth), but the quantity of lymph circulation is greater than in later years. The observations of Kramstyk show that particles of fat are very easily absorbed in early life. Brunner's and Lieberkühn's glands are only partially developed

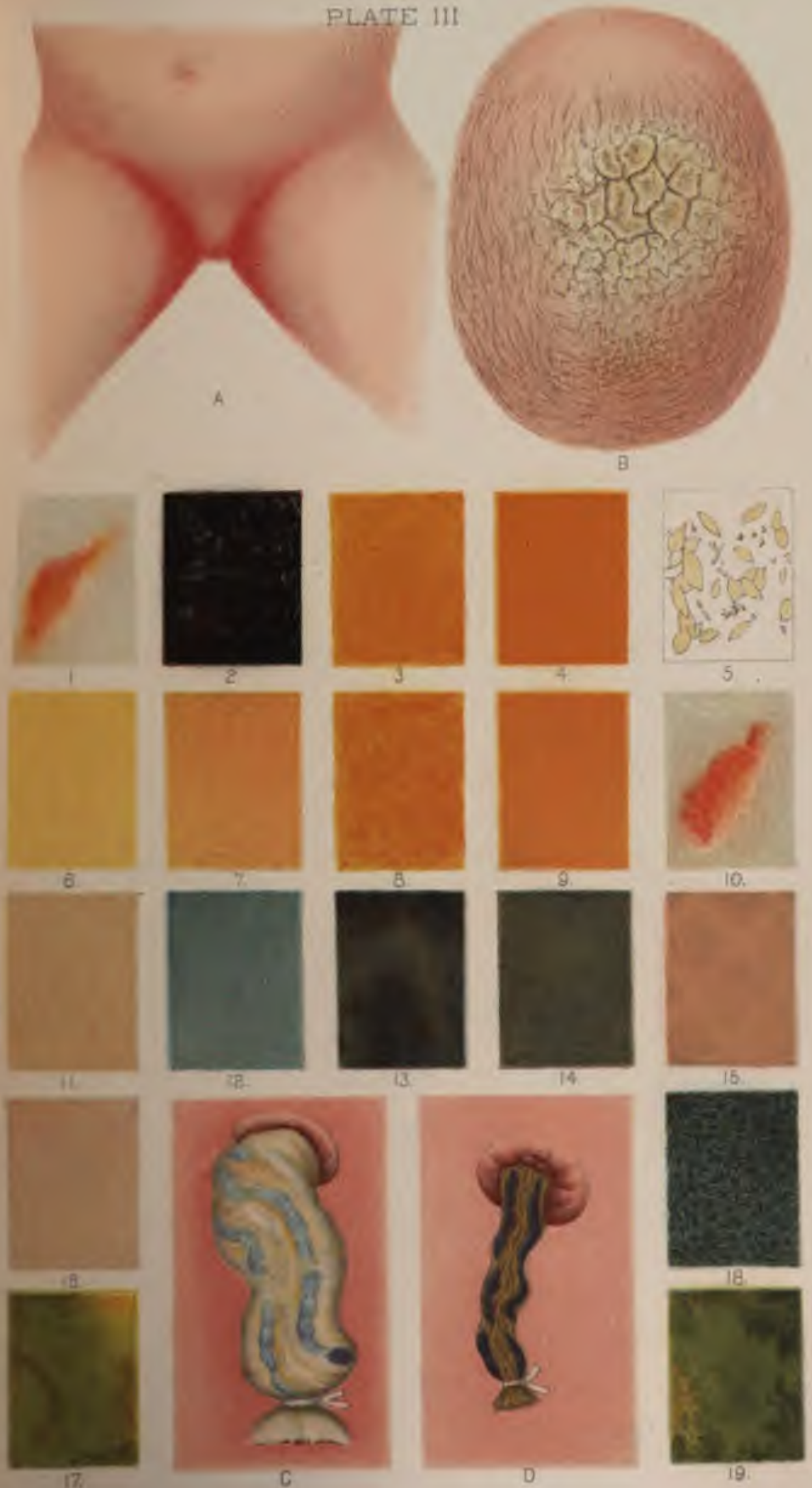
PLATE III.

A. Intertrigo.
B. Seborrhœa capitis of infants.
C. Cord, 24 hours old and 6 days old.

Napkins.

1. Detritus of uric acid infarction (stain on napkin during early days of life).
2. Meconium.
3. Color of fæces resulting from good human breast-milk.
4. “ “ “ “ “ “ “
5. Crystals of uric acid and urate of ammonium (hedgehog crystals) taken from 10.
6. Substitute feeding. Fat, 2 per cent.; milk-sugar, 5 per cent.; proteids, 1 per cent
7. “ “ “ 3 “ “ 6 “ “ 1 “
8. “ “ “ 4 “ “ 7 “ “ 1 “
9. “ “ “ 4 “ “ 7 “ “ 1 “
10. Detritus of uric acid infarction in excess.
11. Bile-stain on napkin.
12. Color of fæces after bismuth 3 grains was given every two hours for six doses.
13. “ “ “ “ 4 “ “ “ “
14. “ “ “ “ was omitted for twenty-four hours.
15. Color on napkin commonly seen, but in this case excessive in amount and pathological from uric acid.
16. Color of “clay-colored” fæces.
17. Color of the change in milk-fed (breast or otherwise) infant's fæces which may occur just before or just after they are passed (not necessarily pathological).
18. Pathological color as seen in case described on page 809.
19. “ “ “ “ “ “ “

PLATE III



in early life; the solitary and agminate follicles are rich in lymphoid tissue.

THYROID.—The thyroid body is relatively greater in the infant than in the adult.

URINE.—The function of the kidney begins quite early in fœtal life, and the bladder has been found to be full of urine at birth. The urine is small in amount at birth, and during the first twenty-four hours it is not uncommon to find little or none passed. The urine which is first passed is usually dark, cloudy, and acid; later it becomes clear, pale straw-yellow, and usually of neutral reaction. Its specific gravity (1.010 at birth) falls in two or three days to 1.003, by about the fifteenth day is found to be 1.006, and rises from this time steadily till puberty. By the end of the first week and throughout childhood the amount of urine passed in twenty-four hours is relatively greater than in adult life. This in early infancy may be due to the preponderance of liquid food, but is in part the result of the infant's more active metabolism, for the urea is also found to be proportionately increased. According to Foster, the presence of uric and oxalic acid in unusual quantities is a frequent characteristic of the urine of children. It is also stated that the phosphates are deficient, being retained in the body for the purpose of building up the osseous system. The chlorides, sulphates, and urinary pigments are less abundant than in the adult. The proportion of salts increases as soon as a mixed diet is given and closely approaches to the normal proportion for adults. Indican is normally absent in breast-fed infants; in others it is usually absent unless there is a disturbance of digestion. The *uric acid infarction*, which has been referred to, and evidences of which may last for two or three weeks, consists of urate of ammonium (hedgehog crystals), amorphous urates mixed with uric acid crystals, and some epithelial cells (Plate III., facing page 84). The variations in the amount of urine which has been computed to be passed during the early days of infancy and childhood are very great, as the amount in all probability depends very largely on the quantity of liquid ingested, and also upon the activity of the skin and bowels. It is well, however, to have some general idea of the normal total amount of the urine at different ages in order to understand better the diseased conditions of the kidney.

The difficulties in accurately measuring the amount of urine excreted by very young infants are such that few positive statements can be made as to the quantity. It is sufficient to say that it is about ninety cubic centimetres (three ounces) a day for the first few days, and then rises in amount very rapidly.

Rietz and Cruse state that during the first few days of life the urine contains more or less albumin, and that this disappears at about the seventh or eighth day. It also frequently happens that the first urine that is passed is cloudy. Sugar is occasionly found in the urine of healthy infants during the first two months.

The following figures, compiled in part by Holt, are the a obtained by combining the results of the investigations of Schab Cruse, Camera, Pollak, Martin-Ruge, Berti, Schiff, Herter, V Renault, and others:

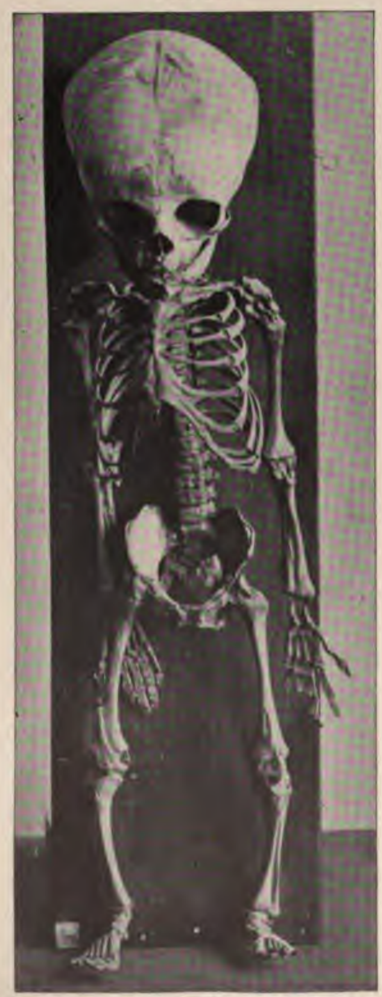
TABLE 22.

		Daily Quantity of Urine in Health.	Specific Gravity.	Ratio of Uric Acid to Urea.	Daily Quantity of of Urea in Gm.	Quantity of Urea per Kil. of Body Weight.	N K
Days	1	0–60 C.C.	1.010–1.012	1 to 14	0.076–0.114	0.0205	
	2	10–90 "					
	3						
	4	90–250 "			0.140–0.660		
	5						
	6		1.004–1.008				
	7						
	8						
	9	150–400 "				0.0919	
	10					0.23	
Months	1				0.90–1.40		
	2		1.004–1.010	1 to 60–80			
	3	210–500 "				0.5 (?)	
	4				3.0 (?)		
	5						
	6						
	7						
	8						
	9	250–600 "	1.006–1.012				
	10						
	11						
	12						
Years	1						
	2						
	3	500–800 "		1 to 50–70	13.09 to 14.01		
	4		1.008–1.016			Boy, 1.017	B
	5					Girl, 0.961	G
	6	600–1200 "				Boy, 1.06	B
	7					" 0.811	
	8					" 0.61	
	9			1 to 45–60	16.05 to 21.03		
	10						
	11	1000–1500 "	1.012–1.020			" 0.88	
	12						
	13					" 0.606	
	14						
Adult						0.55	

The urinary sediment of the young infant shows on micros examination mucus, many epithelial cells, crystals of uric acid, and calcium oxalate; also amorphous urates, occasionally a few casts, and rarely a granular cast. The urine of later infancy an hood has no microscopical peculiarities.

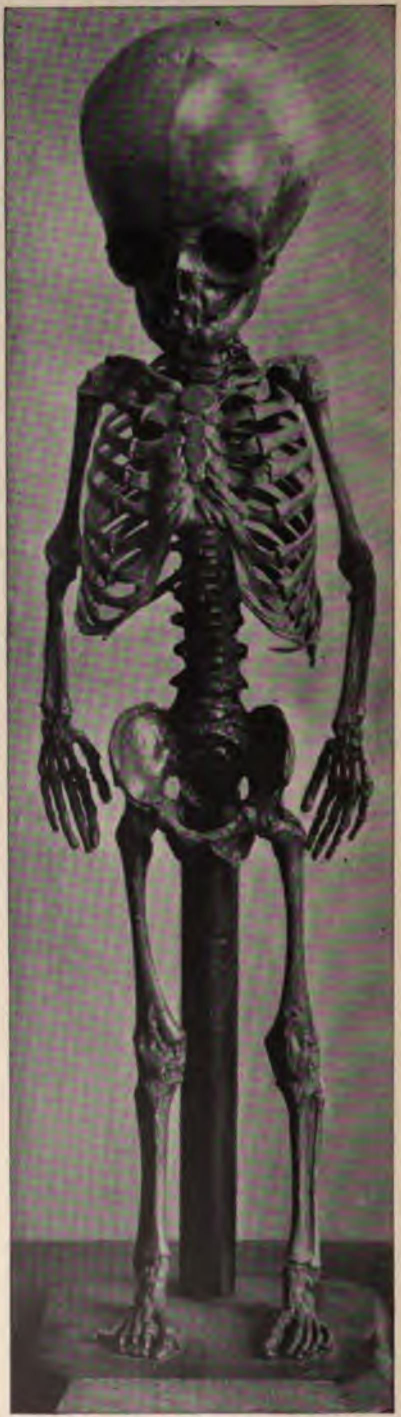
INTESTINAL DISCHARGES.—The contents of the intesti tinue to be mixed with meconium for three or four days or a we longer time being when the infant is weak and does not nurse well. this time the infantile discharges, which have a characteristic app as distinguished from those of the older child, appear. It is neces be familiar with their characteristics, as they are an important guid proper feeding of the infant and are an index showing whether t is properly digested and assimilated. When the nutriment is mil the percentages of its different elements corresponding to what is n found in good average human milk, the discharges are of a golden color, smooth, unformed, of medium consistency, showing a lar portion of water, and sometimes changing on exposure to the

Fig. 28.



Infant at term, showing large head, large anterior fontanelle, small thorax, cartilaginous sternum, tilted pelvis, and bow-legs.

Fig. 29.



Infant at 19 months, showing large head, small anterior fontanelle, ossification of sternum, tilted pelvis, and straight legs.

Warren Museum, Harvard University.

greenish yellow. They as a rule contain undecomposed bile-pigment and bile-salts, while the older child's and the adult's discharges do not contain the bile undecomposed. The amount of fæcal discharge in the first day of life is about forty-five grammes (one and one-half ounces), and increases in the following days to fifty grammes (one and two-thirds ounces). It consists of mucous, fat, epithelial remains, and a slight amount of albuminoid material. In early infancy there are from two to four discharges daily. As the child grows older there are two and finally one in the twenty-four hours. They do not lose their yellow color until amylaceous or albuminous food is given, when the different shades of brown begin to appear. They are not formed until something besides milk is swallowed. Starting at birth with the sterile meconium, infection by the mouth and rectum quickly occurs, and in a short time almost any form of bacteria may be found in the discharges, but chiefly such putrefying forms as proteus vulgaris (Jeffries). With the suckling of the infant and the substitution of the refuse of the milk and the secretion of the digestive tract for the meconium, a sharp transition occurs. Instead of the generally distributed forms, causing decomposition, only two kinds of bacilli are now regularly found, the bacillus lactis aërogenes and Brieger's bacillus, the first chiefly in the upper parts of the intestines, the second in the lower part. When the infant begins to take a mixed diet, quite a number of forms of bacilli appear, among them the streptococcus coli gracilis, the putrefying green fluorescing, a tetrad coccus, and several kinds of yeast. The color of the infantile intestinal discharges when the nutriment is milk alone, whether human or animal, seems to depend somewhat on the percentage of fat, as is seen in Plate III., facing page 84, where the color resulting from the food containing different percentages of fat is represented in Nos. 3, 4, 6, 7, 8, and 9.

INFANTILE SKELETONS.—Fig. 28 represents the skeleton of an infant at term, and Fig. 29 that of an infant at nineteen months. These skeletons illustrate some of the differences which occur in the bones during their process of development.

In the younger subject the head is large in proportion to the small thorax, and there is a lack of development of the face in comparison with the head, which is very evidently due to the rudimentary development of the jaws. The anterior fontanelle is widely open. On examining closely the sternum it will be noticed that it is not in one piece, as in the adult, but that the centres of ossification with the intervening cartilaginous connections are well marked. The pelvis is tilted forward, as compared with the adult's, and is small and contracted. The legs are not straight, as in the older child, but show decided bowing of the tibia and fibula. This characteristic condition of the legs in intra-uterine life is present at birth and continues for some months, the bones usually becoming straight by the time that the period of walking has been reached. In the skeleton of the infant nineteen months old, the legs have developed naturally in their growth and are straight. The pelvis still tilts somewhat, but is evidently less contracted, or rather has begun to enlarge. The thorax has broadened in comparison with the head, and the cartilaginous sternum has become to a large degree bone. The head is still large proportionately to the face, although the jaws have developed considerably beyond what is seen at birth. The anterior fontanelle is quite small in comparison with the fontanelle of the new-born infant. These are the chief characteristics of the infant's and child's skeleton.

NORMALLY DEVELOPED INFANTS.—One of the greatest drawbacks to the proper appreciation of the kind of knowledge which is needed to examine and treat children successfully and intelligently when they are sick, is the lack of precise facts concerning healthy children. It is therefore important to know at a glance whether it is normal for a child not to sit alone or not to stand alone, and to understand its childish actions, whether in creeping or in walking.

The ages at which infants develop so as to gradually become children are important to determine, for it is just as wrong to task by premature use the function of locomotion as that of digestion. A normal infant should hold up its head without support at the second or third month; it should sit alone at from seven to nine months, creep at about ten months, stand at about twelve months, and walk at about fifteen months.

TOPOGRAPHICAL ANATOMY OF THE EARLY PERIODS OF LIFE.—It is important, for purposes of diagnosis, to recognize the fact that the organs differ in the space which they occupy in the body according to the stage of development of the child. Well-marked periods are thus shown to exist by physical examination as well as by anatomical research, and the results of these different methods of investigation are found to correspond. I have always found that a careful consideration of the period of development is of the first importance when beginning to make a diagnosis of disease, especially of the heart and lungs. The large size of the liver in infants and the comparatively greater proportionate size of the heart to the lung in the middle years of childhood are striking instances of the truth of this statement, and should warn us that more than ordinary care should be employed in diagnosticating a pneumonia of the right lower lobe behind in infancy, or a dilated heart in childhood. Three periods of growth are of especial significance in this connection: 1. A period corresponding to the development of the organs in the first year, especially in the first half of the year. 2. A period occurring during the fourth, fifth, sixth, seventh, and perhaps eighth and ninth years. 3. A period embracing the later years of childhood.

Figs. 30 and 31 represent the first period. This infant, seven months old and normally developed, has, outlined in black, the principal points both in front and behind which will be useful to remember when making a physical examination at this age.

In Fig. 30 the plain dark lines have followed the lower margin of the ribs and the outline of the ensiform cartilage and manubrium. To the left of the lower part of the left sternal line is a small curved line. This represents the absolute dulness of the heart. The relative dulness is very slight, and indeed almost imperceptible, even on light percussion over the sternum. This area of dulness can almost be covered by the end of the finger used for percussion. It is bounded by the fourth rib or third interspace above, and is just within the mammillary line. There is very fair resonance under the whole length of the sternum. The interrupted lines represent the upper and lower borders of the liver. There is not much to say about the upper line, but the lower one is interesting and instructive as illustrating the large size of the liver in early infancy, and how little of the stomach, which is here represented by a dotted line between the edge of the liver and the left border of the ribs, is to be reached by percussion. The stomach is, of course, in this infant, empty. When full, it comes out much farther under the edge of the liver. This general idea of its position, however, is very important when we come to consider cases of improper feeding where we have to determine whether we have a dilated stomach to deal with. The broad black line just above the level of the umbilicus marks the transverse colon, which in infancy has a relatively low position. The cæcum, which is marked by a black circle, stands high in the abdomen, near the anterior superior spine of the ilium. The upper piece of the sternum is also outlined, as is the clavicle and first

Fig. 30.

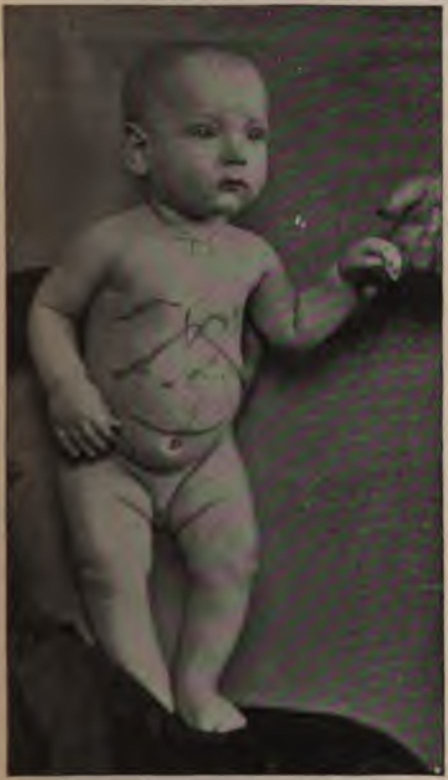
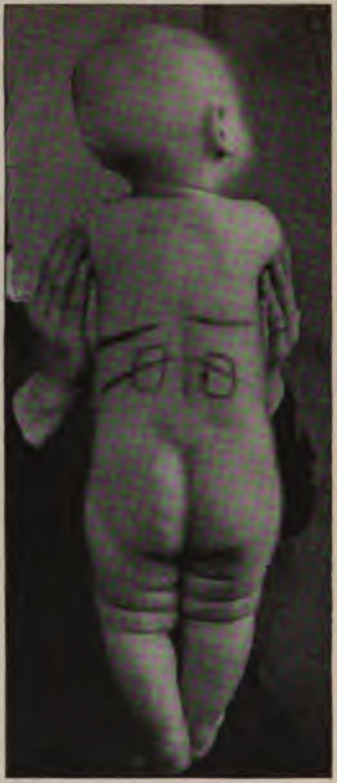


Fig. 31.

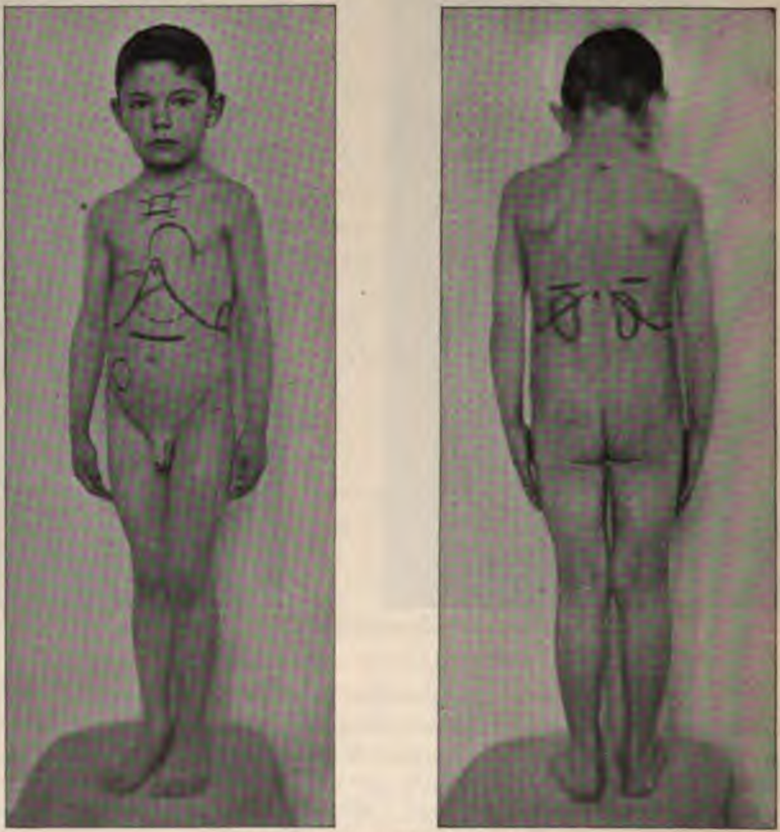


Normal infant seven months old.

rib. Fig. 31 shows the same infant's back; the lower borders of the thorax, the kidneys, and the lower borders of the lungs are outlined. The left kidney is decidedly higher than the right at this age. While the lower border of the lung on the left comes down as far as the tenth rib, the corresponding border of the right lung, owing to the large size of the liver, descends only as far as the ninth rib.

Figs. 32 and 33 represent a child in the second period of its growth. In this middle period of childhood the heart has developed more rapidly proportionately than the lungs, and takes up more space in the anterior portion of the thorax.

FIG. 32. FIG. 33.



Normal development at six years.

He was six years old, properly developed for his age, and presented certain points of interest which differ from the infant and the adult, and which should be carefully taken into account when we are making a physical examination at this age. The manubrium, the clavicles, the first and second ribs, the ensiform cartilage, and the lower borders of the thorax are marked in black. The area of cardiac dulness is far greater than in the infant, Figs. 30 and 31. The dulness should, so far as the sternum is concerned, be determined by light percussion directly over the sternum from above downward. In this way we can detect the change in the percussion note over the lower part of the sternum better than by percussing from the lung to the sternum, since the former is so much more resonant that the sounds are more difficult to distinguish and are often misleading. The upper resonant part of the sternum, on the other hand, presents an excellent opportunity for comparison, and brings out the delicate shades of sound which are needed in getting the relative dulness. This relative dulness, however, is usually pronounced under the lower part of the sternum in this period of development, and it shades off into the absolute dulness of the precordia. Absolute dulness under the sternum, unless depending on pathological conditions, is rare even at this age, when it is also rare not to have this physiological relative dulness. In this period the dulness of the heart extends higher in the left parasternal line than at any other time of life. The lower border of the third rib usually marks the upper border of the absolute dulness, which extends also to the left sternal line and keeps well within the mammillary line. The relative dulness, on the other hand, reaches as high as the lower border of the second rib. It then passes to the right under the upper third of the sternum, descends obliquely to the fourth right costal cartilage, and then keeps closely to the right sternal line.

To the left it extends well out to and perhaps a little over the mammillary line. This is a far different result of percussion from that which is found in the adult, and also in the infant represented in Fig. 30, in whom there is no dulness under the sternum and the absolute dulness rises only as high as the fourth costal cartilage in the left sternal line, and the relative dulness only to the third interspace. The relative dulness also extends only as far as the mammillary line. The impulse of the heart is usually found a little higher in infants and in young children, irrespective of these periods, than in older children and in adults.

A much smaller space is occupied at this age by the liver than in infancy. The liver is indicated by the double line, which rises as high as the fifth rib in the mammillary line, and to the attachment of the sixth or seventh right costal cartilage to the sternum. The dotted line of the stomach, on the other hand, occupies a much larger space than in the infant. The line of the transverse colon stands proportionately higher, the cæcum rather lower. Fig. 33 shows the lower border of the right lung still to be a trifle higher than that of the left, and to come to about the upper border of the tenth rib, while on the left side it extends to the lower border of the same rib. At this age the liver has diminished in size relatively to such an extent that the difference of the position of the lower borders of the lung is but slight.

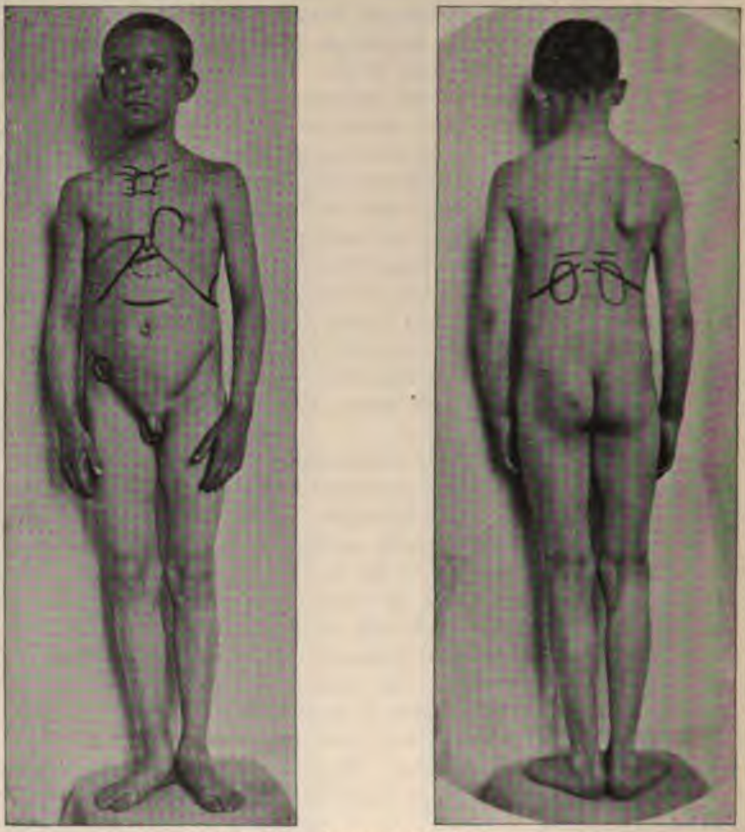
The kidneys are about on a level on both sides. The first and twelfth dorsal vertebræ are also indicated as landmarks. This child is passing through transitional stages of physical development, and is gradually approaching the adult type of perfected growth.

Perfected growth, so far as the topography of the organs is concerned, is reached in the third period represented in the last years of childhood and at about the age of puberty. The organs of the child seem at this age, although they have not yet acquired their complete growth, to present for purposes of percussion the outlines which we are accustomed to see in the adult, with the exception possibly of the position of the cæcum.

Fig. 34 shows a normally developed boy, twelve years of age, and illustrates remarkably well the relative topographical correspondence of later childhood and adult life.

As in the boy of six years, the manubrium, clavicle, first and second ribs, ensiform cartilage, and the lower borders of the thorax are outlined in black. The curved line passing up the left sternal line to the fourth rib and keeping within the mammillary line marks the absolute dulness of the heart, and corresponds to the topography of the adult's heart. The upper line of the liver is about at the level of the fifth rib in the mammillary line, and does not extend beneath the lower border of the ribs, but is just below the tip of the ensiform cartilage. The dotted line represents the stomach. The spleen has its upper border at the ninth rib, and its lower portion comes down as far as the lower border of the eleventh rib. The cæcum is marked in the upper part of the right groin. The transverse colon is about midway between the stomach and the umbilicus. Looking at this same boy from behind (Fig. 35), it will be seen that the kidneys and the lower borders of the lungs are in about the same relative position as occurs in the adult. I have also indicated the first and twelfth dorsal vetebræ.

FIG. 34. FIG. 35.



Normal development at twelve years.

These representatives of the normal development of important periods of life not only were carefully mapped out by myself by percussion and in accordance with the anatomical knowledge which we possess on this subject, but also were verified by Professor Dwight, who examined each child carefully and satisfied himself that my marking was correct.

NURSERY.—Before undertaking to treat the various diseases of early life, it is of great importance to acquire a practical knowledge of the care of the infant and child in health. It is essentially in the nursery that we should study the healthy child, as the nursery is its home, where it feels most at ease and behaves in the most natural manner. The general hygiene of the child is represented in its nursery, and we should therefore by our knowledge and advice so direct these questions of nursery hygiene as to give this sensitive, easily impressionable young human being the best opportunity to develop into a healthy and vigorous adult. We cannot, of course, in every case procure for the child the surroundings which are best for it, but we can at least impress on the parent what these surroundings should be, and how important they are for the general health of the child. The nursery should be high from the ground and out of reach of the dampness which arises towards the latter part of the day.

SUN AND WINDOWS.—It should have a sunny exposure and large windows high enough from the floor to avoid having the younger children continually pressing their faces against the glass to look out, and thus frequently catching cold from the little currents of air which penetrate most window-casings. The mothers often overlook this simple manner of catching cold, and wonder how their children, who are so closely watched, could have contracted the catarrhal conditions which arise in this way.

PAPERS AND CARPETS.—It is much better not to have a paper on the walls or a carpet on the floor. Young children are very susceptible to inhalation poisons, and to organisms of all kinds. Many a case of anæmia, nasopharyngeal catarrh, and stomatitis ulcerosa has apparently arisen from arsenic in the paper. Dust also, with its multitude of organisms, which with the most careful sweeping it is impossible to get rid of, is another source of irritation to the respiratory tract. Even small amounts of arsenic in the contents of the house appear to affect certain children, and the paper itself is a receptacle for micro-organisms which are difficult to eradicate.

PICTURE-MOULDINGS.—It is advisable not to have any picture-mouldings on the walls, as they are a place for dirt to gather which it is impossible to remove properly.

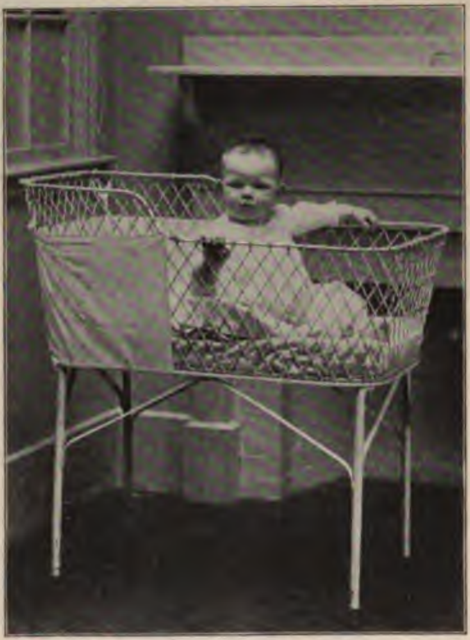
FLOOR.—There should be as few cracks as possible in the floor, and it should be smooth, so as to be easily cleansed. The floor, however, should not be highly polished, for children frequently fall while playing, and sometimes quite severe accidents occur in this way.

WALLS AND CEILING.—The floor, the walls, and the ceiling should be painted. Not only can they then be frequently washed and scrubbed, but when the child happens to have any of the contagious diseases, the whole room can so easily be disinfected that it saves much trouble and expense.

RUGS.—A rug is desirable in the middle of the room. It should never be an antique; in fact, it is better to have new, simple carpet rugs. The rugs should not be too large nor too heavy to be frequently taken out into the open air and thoroughly beaten.

BED.—The child's bed should be iron, painted so that it can be carefully cleansed by wiping, and its sides, as the child grows older, should always be kept high enough, by some simple contrivance, to prevent the child from climbing over them. As few hangings and useless curtains, with which the mother is usually so desirous of draping the bed, should be used as possible. Fig. 36 represents the bed used in the wards of the Infants' Hospital, and it is to be noticed that the bed is of such a height that the infant can be easily handled and examined when sick by the physician without his stooping much.

FIG. 36.



Infant's bed, Infants' Hospital.

Having the bed high from the floor serves another purpose,—namely, that the draughts from the windows and doors pass under the bed rather than on to it.

Pillow and Mattress.—The pillow and mattress should be of felt, folded so as to be soft and comfortable, and pillow and mattress cases should be used. When this is done the felt can be unfolded and aired every day, and both the felt and the cases washed and boiled when necessary. The mattress should be protected by a rubber sheet. Especial precautions should be taken that the child does not kick off the clothes at night. It is well for the nurse's bed not to be close to that of the child. This entails a little extra trouble on the nurse's part, but her breath is not a healthy pabulum for the child's lungs, which require fresh, pure air of their own.

CLOSETS AND DRAWERS.—The child should have its own closet and its own bureau-drawers. The nurse's belongings ought to be kept in a separate room. The closets and drawers should be cleansed at least once a week.

FURNITURE.—There should be sufficient furniture in the room for comfort, but stuffed furniture should be avoided. As little as possible that is complicated or cumbersome should be kept in the child's nursery.

CURTAINS.—Only simple muslin curtains, which can be washed, should be used at the windows.

TOYS.—As a child puts everything that it gets hold of into its mouth, care should be taken not to allow it to have toys with colors that can be soaked off by its saliva, which would perhaps poison it. Toys also which are made of woollen materials or of feathers should be avoided, as particles easily come off them.

HEATING AND VENTILATION.—The heating and ventilation of the nursery are of great importance. The child requires pure, warm air. The temperature of the room can vary somewhat according to the climate, but, as a rule, the average should be from 18.8° to 21.1° C. (66° to 70° F.). The open wood-fire is best both for the character of the heat which it gives, and for its value as a means for promoting ventilation.

A plain piece of wood the width of the window, about 10 cm. (4 inches) high, and made to fit closely to the window-sill, is the best ventilator, but is rarely needed where a wood-fire is burning in the room. The upper sash can also be lowered for a few inches if more air is needed.

DRAUGHTS.—We must take into consideration the currents of air in the nursery, so that the mother, understanding the atmospheric conditions which surround her child, can give the simple directions, which she has learned from us, to the nurse. This is by no means an unnecessary precaution, for one of the worst cases of rheumatism in the hip-joints which has come under my notice was that of a child two years old who was allowed to sit on the floor with its back to the open door, and directly in a line with the open fireplace. The direction of the currents of air between the doors, windows, and open fireplace is admirably and scientifically described by Mr. John Pickering Putnam in his valuable work entitled "The Open Fireplace," in which the direction of the cold-air currents is shown to enter the windows, descend to within a few inches of the floor, and then to radiate towards the open fireplace and doors. If the child is much on the floor, a sheet can be placed over the cracks of the door; and plain white sheets are always the best articles for screens or portières.

Where the current of air is too strong it can be tempered by pinning a towel across the opening between the upper and the lower sash. This should at once suggest to the mother that parts of the room, on account of these currents of air which from doors and windows pass over the floor to the fireplace, should be avoided not only for bathing but also for playing on the floor.

A high *fender* covering the entire opening of the fireplace, and fastened so that the older child in playing cannot pull it down, is an important part of the nursery equipment. It answers two purposes,—one to prevent the sparks from flying out on the child, the other to prevent the child from falling into the fire. Serious accidents have happened from a lack of proper precaution regarding this apparently self-evident necessity. The hot air from the fire radiates in all directions into the room.

SCALES.—Properly adjusted scales are an important part of the nursery equipment. The scales which are usually provided are, as a rule, very inadequate for the minute and daily weighing, the results of which are at times of such great assistance to the physician in the management of the infant's food. Never hang an infant in anything on a hook to weigh it. Such weights are usually, from the continual kicking of the infant, quite incorrect. Do not think that the kitchen grocery scale is good enough for the infant. We can afford to have incorrect and approximate grocery weights, but cannot afford to apply these methods to the growing infant, with its unstable equilibrium. The scales should be of a small but solid platform variety, which can be placed on a firm table by the tub where the infant is to be bathed, for use before the bath.

The scales should weigh as low as four or five grammes (one drachm). A basket, with a small soft blanket lining it, is placed on the platform of the scale, and the naked infant is weighed in the basket. The scale is balanced, and the infant immediately taken out of the basket without stopping to read the weight, so as not to expose it too long while uncovered. When the infant has been dressed the scale can be read, and the balance-weight minus the weight of the basket and blanket (which can, of course, always be a constant quantity) gives us the exact weight. Weighing with the clothes on is a very unsatisfactory procedure.

BATHING.—The question of the bath is of a good deal of importance in the early months of life. Unless there is some definite contra-indication, an infant should be bathed every morning. The contra-indications are if the skin or nails turn blue, or if the infant seems in any way to show symptoms of weakness or lowered vitality after bathing, such as are represented by cold extremities and nose, or an unusually quickened respiration. In these cases sponging, merely sufficient for cleanliness, is to be substituted for the bath. The bathing should be done with celerity, the tub being placed on the side of the fireplace opposite from the window, and fronting the latter, so as to avoid draughts and insure a good light. The nurse should sit with her face to the light and have the infant on her lap, wrapped up in a warm blanket, with its feet towards the fireplace, and its head in such a position as regards the window as to avoid having too much light in its eyes. There should be a rack for the towels, which should be kept warm in front of the fire while the infant is being bathed. The clothes should in like manner be neatly spread out on another rack, ready to be put on as soon as the infant has been dried.

TEMPERATURE OF BATH.—The water should vary in its temperature somewhat with the age of the infant, but should never be so cold as to cause blueness or cold extremities. We must also be careful not to have the water too hot, as this has sometimes proved to be injurious. Each infant, however, must have the temperature of its bath adapted to its own vitality. A convenient bath thermometer is one which is guarded from breaking by a wooden frame, which also allows it to float in the water, and the nurse is thus enabled to see at a glance that the bath-water is remaining at the proper temperature. The following table may be taken as a guide:

TABLE 23.

Temperature of the Bath for Different Ages.

Age.	Centigrade.	Fahrenheit.
At birth	36.6°	98°
During first three or four weeks	35°	95°
One to six months	34°	93.2°
From six to twelve months	32.2°	90°
Twelve to twenty-four months	30°	86°
Then gradually reduce in summer to	26.6°	80°
In the third or fourth year, if possible, reduce to	23.8°	75°

The nurse should first wash the face in clear water, keeping the body and limbs wrapped up in a warm blanket. The face is then wiped with a soft towel. She should gently cleanse the nose, the corners of the eyes, and the external ears. The nose is especially important, for the infant's vitality is easily affected by occluded nares. The nurse should then soap, wash off, and dry the scalp. The sponge and water in the other division of the bathing basin are then used for soaping the body and extremities. Especial care should be paid to the folds of the neck, the axillæ, groins, genitals, and anus. The temperature of the water in the basin and bath should be taken from time to time with the wooden bath thermometer until the washing is over. The proper warmth of the water is to be kept by adding when necessary a little hot or cold water from cans within easy reach.

TUB.—The tub, which is preferably made of rubber hung on a simple wooden frame and sufficiently high to prevent needless stooping on the part of the nurse, is placed on the nurse's left, at a convenient distance from her chair.

BASIN.—In front of the nurse is the double washing basin, which is merely a china basin divided into two compartments, and fitted to a wicker stand, also sufficiently high to prevent the nurse from stooping as she uses it. To the right of the nurse is the table, with the scales on one end and the toilet basket on the end towards her.

SOAP.—The soap should be white castile, or any kind which is free from irritating elements.

SPONGES.—There should be two sponges: one goes in one side of the washing basin, and is for the head and face; the other is to be used in the opposite side of the basin, and is for the body and extremities. The body and limbs having been thoroughly and quickly soaped, the nurse should gently lower the infant with its face up into the clear water in the bath, being careful not to frighten it or to drop it. This is not an unnecessary warning. I have known infants, even in the hands of ordinarily careful mothers, to be dropped from the bath or scales, with a resulting permanent injury of the spine or hip. After allowing the infant to kick and splash for a few seconds, it is taken back into the nurse's lap and carefully dried with a warm, soft towel. Never soap and wash the infant in the bath, but always on the lap.

POWDER.—When the skin is perfectly soft, clear, and in a normal condition, no powder is needed. Where there is any slight irritation, which, at times, is liable to occur when the skin has not been kept sufficiently dry, especially if there is a decided redness in the folds of the skin, as of the neck, axillæ, or groins, the following powder can be applied:

PRESCRIPTION 1.

	Metric.	Gramma.			*Apothecary.*	
℞	Pulv. zinci oxidi.............	7	5	℞	Pulv. zinci oxidi	ʒii;
	Pulv. amyli trit.............	60	0		Pulv. amyli trit................	℥ii.
	M.				M.	

No perfume of any kind should be added to the powder. The infant should be sweet and pure in itself, without accessory odors.

CLOTHING.—It is very important that those who care for the infant should not only clothe it properly but should understand why one method of clothing is better than another. The surface of the infant's body is greater in proportion to its entire weight than is the case in the older and hence larger human being. Greater surface means that there is a greater opportunity for radiation, and hence that the smaller subject will cool off more quickly, other conditions being equal, than the larger one. We therefore see at once that much care should be given to the question of warmth in the infant. Any exposure of the body or limbs in either infants or children is unwise. A very important factor in the problem of growth in the infant is perfect freedom of motion for its legs and arms and for the respiratory and abdominal muscles. It should also be thoroughly understood that pressure on any portion of the body or limbs must produce evil results, by displacing organs which should be allowed to have entire freedom of position in their respective cavities.

Too litttle warmth will do harm, by preventing the proper metabolism of the tissues and thus reducing the animal heat. Too great warmth, on the other hand, by causing inequalities in the circulation, will in like manner be detrimental to the child's growth and vigor. Clothes which bind any part of the infant tightly cannot but press out of their natural position whatever happens to be beneath the point of pressure, whether it be the liver, the intestines, or the toes. The clothes, then, must evidently be warm and loose, and we must bear in mind that loose clothes are warmer than tight ones, from the very fact that they do not interfere with the natural activity of the circulation, and that they give freer play to all the muscles which produce the normal warmth arising from exercise. We must remember that the only way in which the infant can obtain the exercise so much needed for proper growth, and which is so easily obtained by the older child in running about, is by continually moving its legs and arms and thus accelerating the muscular action of its thorax and abdomen.

An important item in the proper management of the infant in its nursery is that it should be irritated as little as possible by unnecessary delay in dressing it after its bath. Useless stitches, buttons, and articles of clothing should be dispensed with, and a method adopted which, while combining the necessities of dress, will allow the dressing to be finished before it has time to annoy the infant.

ABDOMINAL BAND.—There is no necessity for using beyond the first two or three weeks the usual flannel band supposed to be so indispensable by the average nurse. Herniæ, whether umbilical or inguinal, cannot be obviated, and in fact may be produced, by undue abdominal pressure.

The abdominal band shown in Fig. 37 A, made of light, soft flannel, can be smoothly applied over the dressing of the cord and kept in place with moderate pressure by means of safety-pins.

The band can soon be replaced by a somewhat elastic knitted garment (Fig. 37 E, A), half band and half shirt, with shoulder-straps of the same material to hold it in place, and a tab in front to fasten it with a safety-pin to the napkin (Fig. 37 E, B).

This shirt can be made of soft wool or silk, or, as I have recently found, can be knitted in any form or size from half cotton and half silk.

This knit material can also be used for the undershirts (Fig. 37 B and Fig. 38 F, pages 100 and 103). Garments made in this way are the best that I have ever seen. They are warm, soft, and delicate, have no seams, can be washed without shrinking, and retain their elasticity much better than those made from other materials.

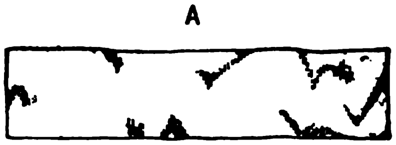
NAPKINS.—The napkin (Fig. 37 E, B, page 100) is folded and fastened with safety-pins as is customary for keeping it in place. The usual napkin is very cumbersome and heavy, besides being expensive. It can be replaced by rolls of soft absorbent gauze, which absorb the urine from the skin, an important quality in cases where the skin is easily irritated. These napkins can simply be cut from the roll, which is kept in the nursery, and, when removed from the infant after a movement of the bowels, can be burned, thus avoiding the trials resulting from the objections of the nurse or the laundress to washing the napkins. If, however, the mother prefers the regular old-fashioned napkin, small squares of this gauze can be placed in the middle of the napkin, and this will in great measure obviate the more disagreeable part of the napkin-washing, as the square of gauze will hold most of the movement and can at once be burned.

The infant while in long clothes need not have any further covering for its legs, and need have nothing on its feet. There is no particular objection to little knit socks if the mother wishes to use them.

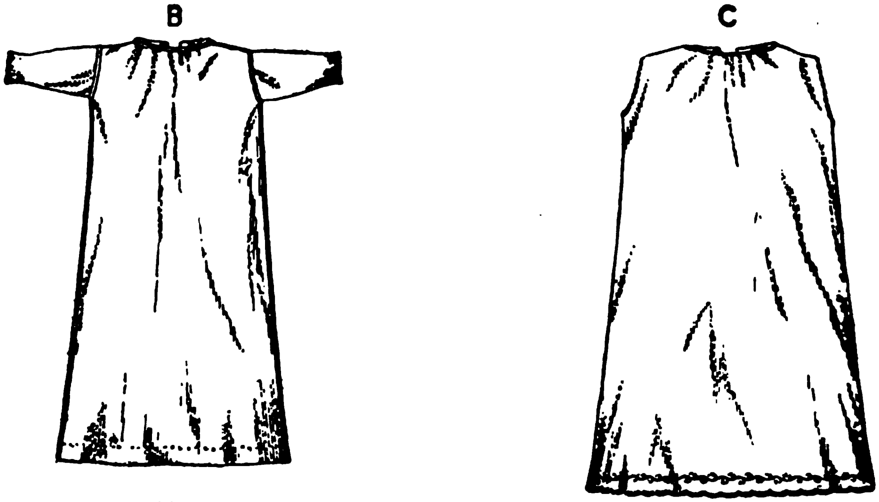
After the nurse has put on the band and the napkin there are left three garments which are usually the clothes needed to complete the infant's outfit of *long clothes*.

Fig. 37.

(*Long Clothes.*)

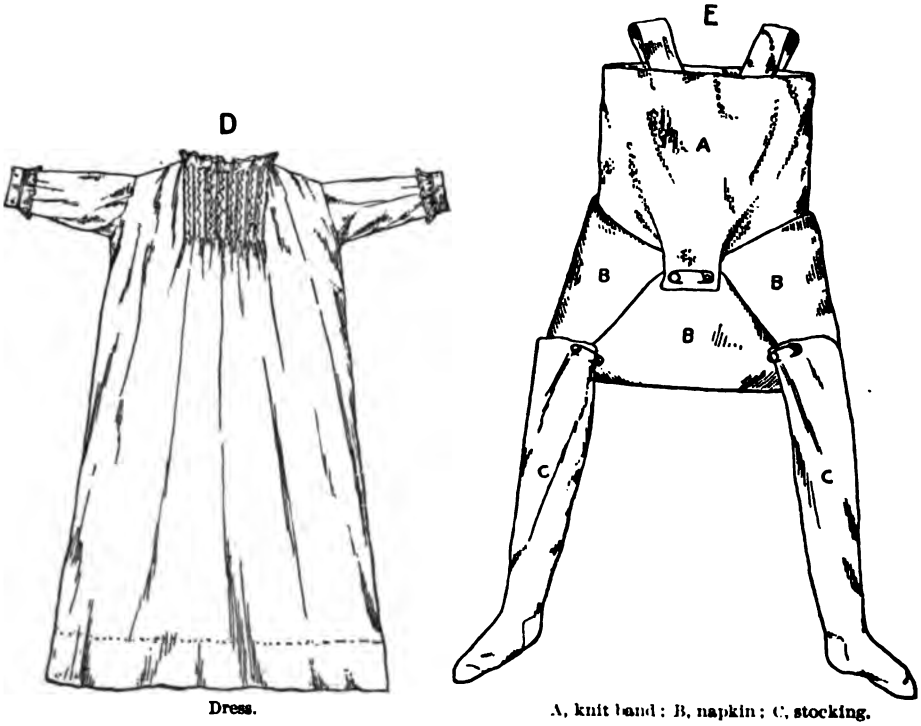


Flannel band for early weeks.



Shirt.

Petticoat.



Dress.

A, knit band; B, napkin; C, stocking.

These garments are the shirt (Fig. 37 B, page 100), the petticoat (Fig. 37 C), and the dress (Fig. 37 D).

Shirt (Fig. 37 B).—The shirt is a garment with long sleeves and high neck, cut almost as long as the outside white slip or dress. Unless it is knitted it is well to have it made of some soft, fine, all-wool material, with the seams finished on the outside to prevent irritation of the skin. It is made to button in the back. A fresh garment of this kind is also sufficient for the infant's dress at night, except during the early weeks of life.

Petticoat (Fig. 37 C).—A flannel shirt cut all in one piece, as the shirt is, made of fine flannel, with no sleeves and with low neck, represents the petticoat. It should be made large enough to go over the shirt, should be of the same length as the dress, and should also be made to button in the back. The taste of the mother can be gratified by any reasonable degree of embroidery which she may wish to put on this second garment, but the shirt should be perfectly plain.

Dress (Fig. 37 D).—The outer garment should be made of some soft white material, such as nainsook, should be large enough to go over the shirt and petticoat, should not be starched, and is usually about one yard long from the neck to the bottom of the skirt. It should have high neck and long sleeves, and should button behind.

The advantage of this costume is that it is loose but warm, and that the three pieces which constitute it can be put on together, the infant having to be turned over only once before the clothes are buttoned. The other methods of clothing usually necessitate turning the baby over several times in the process of dressing.

Before the infant has had its bath, these three articles of dress are to be arranged one inside the other, ready to be slipped on all three at once. This can be done with great celerity, and the dressing process can thus be gone through without the usual accompaniment of irritated cries which are so frequently heard in the nursery, and which are to be deprecated.

When the infant is old enough to have its long clothes changed to short ones, which is at about the time when it learns to creep, the under-garment can be replaced by a knitted or fine all-wool undershirt with high neck and long sleeves (Fig. 38 F, page 103) made short, with an additional white petticoat in winter if desired. The infant should now also have its feet and legs covered with long white wool stockings, which are kept in position by being pinned to the napkin (Fig. 37 E, b, page 100). When the child begins to walk, soft kid shoes should be used with the soles adapted to the natural curves of its feet, as explained on pages 104 and 105.

Stockings.—A word in regard to the stockings is especially needed in reference to the older child in its third, fourth, and fifth years. It is a mistake to think that if we keep the feet and abdomen warm the legs can be left uncovered with impunity. Short stockings and bare legs, in my opinion, should be abolished, as a prolific source of catarrhal conditions. The argument is a poor one that certain children have been known to grow up well and strong with uncovered legs, or even that our ancestors were in the habit of depriving their children of suitable coverings for their necks and arms as well as legs, while they themselves were warmly clothed from head to foot. Our ancestors did and said many things which, to us, convict them of great ignorance. The stockings should be white. This is to insure freedom from poisonous dyes, which at times seriously affect the delicate skin of the young child. Colored stockings are a source of great gratification to lazy nurses and to those who wish to lessen the size of their laundry.

There are three garments which are usually put over the shirt and are considered to complete the short clothes. These are the flannel petticoat, the white petticoat, and the dress, and they are to be made large enough to fit one over the other and thus to be put on all at once.

FLANNEL PETTICOAT (Fig. 38 G, page 103).—The inner garment next to the shirt has a flannel skirt, a cotton waist, low neck, no sleeves, and is fastened with buttons in the back.

WHITE PETTICOAT (Fig. 38 H, page 103).—Next to the flannel petticoat comes a garment with a skirt of some soft white material, with a cotton waist, low neck, no sleeves, and also buttoned in the back.

DRESS (Fig. 38 I, page 103).—Finally, over all the other garments comes the dress, which is made with high neck and long sleeves, and is buttoned behind.

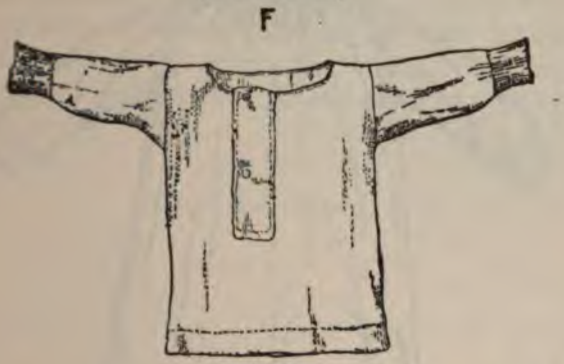
NIGHT-DRESS (Fig. 38 J, page 104).—A regular night-dress can now be used, made of soft flannel, with high neck and long sleeves, and buttoned behind. An extra garment can in cold weather be worn under the night-dress if deemed advisable for the especial child.

FEET.—In young children, although the foot may be well formed, it is very weak, so that the arch is easily broken down. The pad of fat, to which reference has been made under development, is a physiological protection against such breaking down. Children should not be allowed to walk until some time after they are ready to do so, always allowing, of course, that if they insist on walking they can seldom be restrained from doing so. As they get older, long walks with their parents should be forbidden, for it is through these long walks that the evils which have been explained are brought about. The child will get exercise enough at its play, and in doing so will not overtax the arch of the foot, or use its feet beyond the degree which nature intended. Children should not be told to turn the toes out too much, as this puts the arch in a position where the muscles give it least support. The average dancing-school master is a fair example of what over-zealous ignorance combined with the respected traditions of the past can do to children's feet.

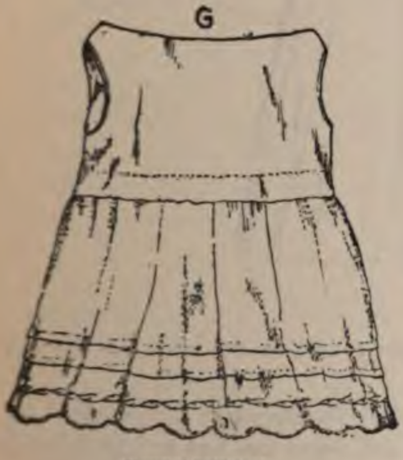
SHOES.—Children's shoes should be rights and lefts, like those of adults, as the present style of straight shoes gives no support to the arch during a very important period of its growth; this, moreover, also tends to push the great toe towards the median line of the foot, so as to cause

Fig. 38.

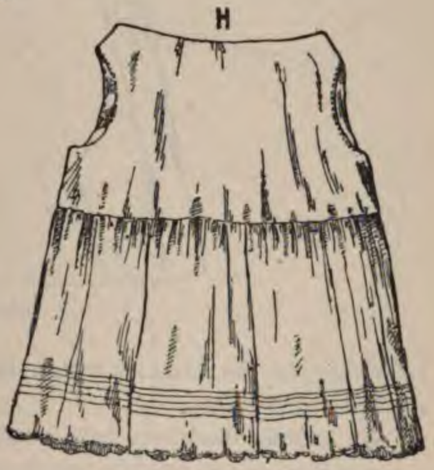
(*Short Clothes.*)



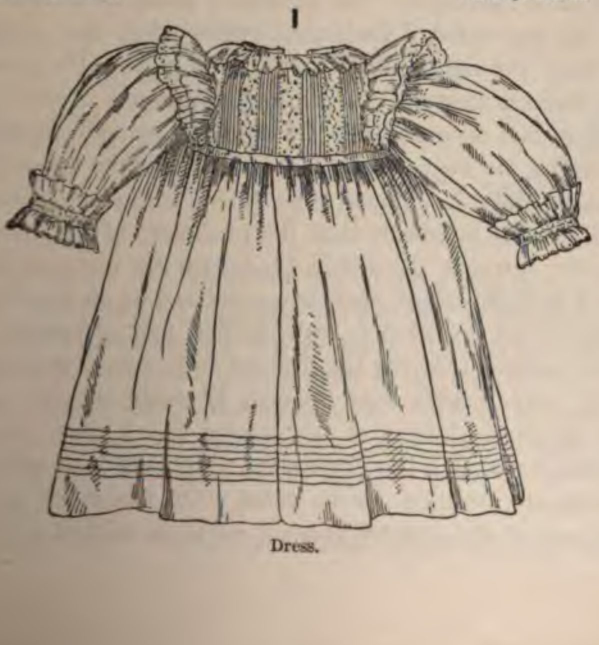
Shirt.



Flannel petticoat.



White petticoat.



Dress.

Night-dress.

enfeebling of the muscles which have so much to do with the proper elasticity of the feet.

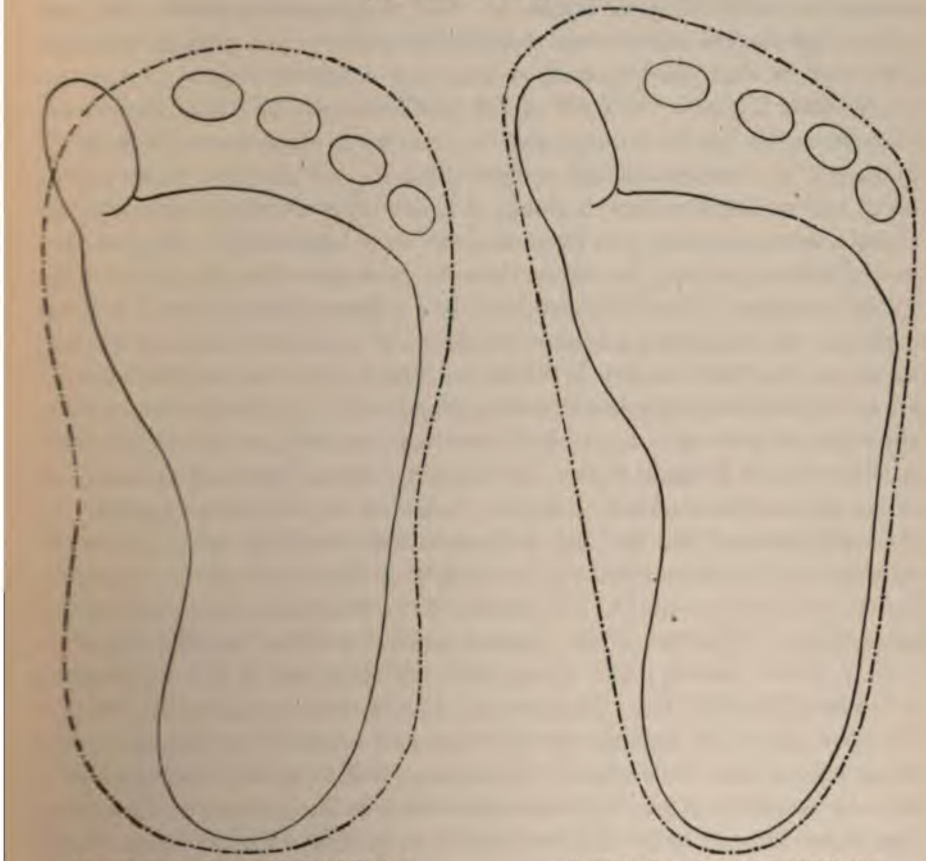
We should, therefore, have shoes properly adapted to the child's foot, —shoes that will at once be comfortable and leave the feet free to develop and fulfil all their functions. The children's shoes as we find them in the stores have the two sides of each shoe symmetrical and equidistant from the middle line; the right and left are told only from the arrangement of the buttons, and are frequently worn interchangeably. Now, the foot has no such median line on each side of which the parts are equally disposed; and its two edges are very different, as a glance at the soles of a one-and-a-half-year-old child's feet shows (Fig. 39).

It is well to note especially that the phalanges of the great toe do not naturally point towards the outer border of the foot: such a position, common as it is in the adult, must be considered as an acquired deformity which started, in all probability, with the first pair of leather boots.

That this matter of forcing the first toe out of its normal position may bring with it very serious consequences is easily shown: as it inclines against the terminal phalanx of the second toe, it often crowds it backward, and finally makes it the distressing "hammer toe," which may even require a surgical operation for its relief. On the inside of the foot, as soon as the axis of the first toe is bent, we begin to find a bulging out of the metatarsophalangeal joint, which in later years, fostered by pair after pair of tight and ill-fitting boots, is capable of giving the most exquisite pain. Still more subtle in its working than this is the trouble that often comes from disabling the great toe from performing its full function. The elasticity of one's step depends largely upon one's power to press down firmly with the great toe and then raise the weight of the body over it as a support; when this is lost by crippling the toe with ill-shaped boots, the muscles not only of the first digit but of many adjacent groups begin to atrophy. This soon leaves the internal arch of the foot without sufficient support, and the long series of woes incident to "flat-foot" is started upon. Therefore, for one and all of these reasons, let us demand that children's feet shall have at least the chance to develop properly in well-fitting anatomical shoes.

FIG. 39.

(Natural size, 1½ years.)



Unsuitable shape for sole of child's shoe. Suitable shape for sole of child's shoe.

SLEEP.—Infants and young children vary much as to the amount of sleep which they need and take during the day. At first they sleep almost continuously, especially if they happen to be somewhat premature. In a few weeks, however, they begin to have regular periods of rest, consisting of several hours' sleep, at first twice in the day, and later once. The more sleep they can be induced to take in the twenty-four hours, the better. As they grow older the amount of sleep which they take grows less, but in the first four or five years of life it is well to try to induce the child to rest quietly on its bed for at least an hour during the day. The number of hours that an infant or child sleeps varies so with the individual that precise rules cannot practically be given; one simple rule holds true,—allow each child to sleep as long as it naturally can.

WHEN TO GO OUT OF THE HOUSE.—If the infant happens to be born in the winter months and the weather is at all severe, it is better to keep it in a well-ventilated nursery than to run the risk of its vitality being lowered by exposure to cold. I believe that infants in our northern climate are exposed to cold far more than they ought to be, and that they need fresh, warm, dry air, rather than the cold and often damp air of our winter months. When they are born in a milder climate, or at a warmer season of the year, they can after the first few weeks be taken out in their carriages often twice a day. When the infant is five or six months old I am in the habit of giving the following directions to the mother as to when she shall send it out. I explain to her that it makes as much difference whether the air is damp or dry, and what the rate of the wind may happen to be, as does the number of degrees indicated on the thermometer. If the sun is shining, the air dry, and there is no wind, the infant can without harm go out for an hour in the middle of the day even at a temperature of —6.6° to —3.8° C. (20° to 25° F.). Where, on the contrary, the air is damp, or the rate of the wind is great, it is better for the infant to remain in its nursery, and, at any rate, not to go out, if the temperature is below 0° C. (32° F.). The practice of allowing the infant to sleep in the open air in its carriage in every kind of weather is, I believe, a bad one; but on the days when it is proper for it to go out, such as I have already described, it can without harm sleep in the open air. The nurse should be directed to protect the infant's eyes from the direct rays of the sun, and not to allow a strong wind to blow in its face.

Where the weather has been too severe or damp for the infant to go out in its carriage for some time, it is advisable to have it dressed warmly and wheeled up and down in its nursery with the window open for fifteen or twenty minutes. To avoid too much draught, blankets can be placed over the cracks of the doors and the open fireplace while the infant is breathing the fresh air. The room being far above the ground, the dampness is avoided, and even a considerable velocity of the wind outside the house will in this way be unable to affect the air of the room, and will not make too strong a draught. Where it is possible, as is the case in certain families who can afford such a luxury for their child, a solarium at the top of the house, especially in cities, is desirable and practical. Not only should an injudicious administration of cold air be avoided, but extreme care also should be taken in hot weather that the child is not exposed to too great direct heat from the sun, and it should never be kept in a hot atmosphere where currents of fresh air cannot have access.

NURSERY-MAIDS.—The idea that the child should be taken care of by an old, experienced nurse is a vicious one. The experience of nurses, as a rule, is that of ignorance rather than of intelligence. Every mother, as she is presumably more intelligent than the nurse whom she employs, and is surely more interested in the welfare of her child, should personally supervise and unhesitatingly investigate all that the nurse does to the child. The nurse's ideas as to what is needed for the child's hygienic surroundings, food, and clothing can well be dispensed with. The mother, learning from the physician what is best for her child, should give her directions to the nurse and see that these directions are strictly carried out. A nurse between the ages of twenty and thirty-five is preferable to one who is younger or older. She should be neat, healthy, strong, cheerful, gentle, and patient. She should be willing to refer small details of the nursery routine to the mother, as well as those which appear of greater importance. The chief attributes of a good child's nurse, in my opinion, are a desire to obey implicitly the orders which she receives from her mistress, and a temperament in harmony with the sensitive nervous organization of her charge. In certain cities, such as New York, Philadelphia, Buffalo, and Boston, schools have been established for nursery-maids. An admirable school of this kind has been carried on for some years at the Infants' Hospital in Boston, where the nurses are trained to be servants as well as nurses, the course lasting for one year. When intelligent girls trained in this way can be obtained, the preferable age is between twenty and thirty-five years.

MOUTH.—A protest should be made against the way in which the nurse, and in fact almost every one who comes near the infant, put their fingers into its mouth on all occasions. It would seem as though the infant's mouth was considered by those who ought to know better as something which was especially made to be felt. Infants are much more likely to have various diseases in their mouths than are adults, and probably one reason for this is that dirt of all kinds is constantly being introduced into them. The fingers should always be thoroughly washed before entering the infant's mouth, and yet unwashed fingers are continually feeling the baby's gums to ascertain if a tooth can be found.

The nurse should be instructed that she is never to kiss the infant on its mouth, or allow any one else to. The germs of disease can well be transmitted in this way. It is partly through ignorance of doing harm, and partly through timidity on the part of the mother in prohibiting it, that a stop is not at once put to this bad habit of nurses and friends, and it is the physician's duty to warn mothers on this apparently trivial but really important question, and to tell them how certain infectious diseases, especially tuberculosis, can be transmitted in this way.

SCHOOL.—Much ignorance of the child's nervous organization is shown by those who should best know how to care for it, at a period of life when its hygienic surroundings, both mental and physical, are extremely important. No one system is good for all children. I am sure that I have seen the kindergarten system do harm to a number of children, although it seems to suit others. Each child should be gauged for itself, and not be forced into any general system, even if that system has proved to be good for the many. No time is lost, in my opinion, in sending children to school at a somewhat later age than is usually supposed to be necessary. I am continually having to take little children out of school who are fretful and have loss of appetite. Neither parents nor teacher seem to appreciate that the little, actively growing brain is overtaxed by too great stimulation and is protesting against such treatment by these general symptoms. Many a child is being dosed with tonics who merely needs rest from school. The parents should keep the most rigid supervision over their children while at school, and notice from their behavior whether they are mentally tired. This supervision should not be left to the teachers alone, however interested they may be in their little pupils. It seems hardly necessary to state that the school-room should be well ventilated, and that at stated intervals during the school hours the windows should be thrown open and the atmosphere of the room completely changed. This should not, however, be done with the children in the room. Attention should be paid not only to what the children eat at lunch, but to how and where the lunch is eaten. A child really needs nothing but dry bread and milk or water between its meals, so far as its nutrition and digestion are concerned.

DEFECTS OF POSTURE.—We can best appreciate the importance of following nature as closely as possible in its methods of developing young human beings so as to perfect their various functions to the fullest extent by examining carefully a group of malformed children.

Back.—The extreme flexibility and slow development of the spine clearly point out to us that nature intends to leave its function in abeyance and bring it into use slowly. If the young infant is allowed to sit or stand at too early an age, the superincumbent weight of the large head tends at once to exaggerate the physiological curves of the spine to a point where they may become pathological.

During the first year of life the strength of the spinal column is slowly increasing. Not before the seventh or eighth month has it acquired sufficient rigidity to warrant the child's being allowed to sit up. Artificial methods, therefore, of making the young infant assume a sitting posture at a period of development when the spine should be comparatively straight should be deprecated. I have met with numerous instances where both parents and nurses were anxious to have the infants, at a very early age, sit for quite a long time strapped in small chairs. In like manner the same infants were encouraged to stand and walk long before the apparatus for locomotion was ready for use. One may ask, how many of these individuals developed a spinal curvature in later childhood? Possibly the risk in a perfectly healthy child may be small. One often, however, in early infancy, cannot determine which individual may become rhachitic, and where rhachitis is present the tendency to abnormal curvature is well known.

We should, then, in our advice as to the proper physical management of the early years of life, be guided by our knowledge of the normal average development. Free play for the infant's legs, when lying on its back in bed, should be a point to be noticed and considered, since we know that pressing down the legs causes strain and curvature in the lower spine. Knowing the great lateral flexibility of the infant's spine, we should advise the nurse not to hold the infant continually on one side. Symmetry of development and free opportunity for natural movement should be our aim in the management of the infant from the very earliest period of its existence. Our knowledge of the great flexibility of the growing spine provides us at once with a most valuable means for treating lateral curvature in childhood, and we are continually seeing the benefit of encouraging the promotion of elasticity by moderate pressure and bending. A case which was under my observation at the Infants' Hospital beautifully illustrates the truth of what has just been said.

A feeble, rhachitic child, nineteen months old, was presented for treatment with a marked lateral curvature in the dorsal region, the convexity being towards the right, combined with decided rotation, following the type of the worst adult cases.

The condition seemed to be purely the result of habit, the patient having been made, when very young, to sit up beyond the limit of endurance of the still undeveloped bones and ligaments. The treatment was based entirely on the elasticity of the spine, and consisted simply of manipulation and recumbency, resulting in a very great degree of improvement both as to the curvature and the twisting.

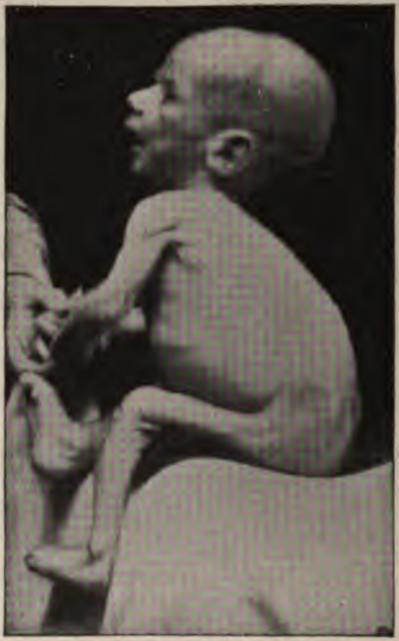
The improper treatment of the young subject's spine, as in infants, for instance, where they are carried altogether on one side, is well recognized as an important factor in the etiology of rotary lateral curvature.

Remembering what has been said regarding the ossification of the different parts of the spinal column, it will be understood that so long as an infant can be made happy in the prone position, whether in its nursery or in its carriage, it will be better for it to be kept in this position, always protecting the eyes when out in the open air from the strong light, and the face from the wind. During the first year when it begins to sit up in its carriage its back should be carefully supported by a pillow.

Fig. 40 represents the harm which can be done by encouraging children to sit up before their spinal columns are sufficiently strong. The infant, six months old, had been made to sit in a chair for hours at a time, strapped in a position which allowed it to use its arms, but such as to render it impossible to fall back and rest itself.

The exaggerated curve of its back corresponds to that which would be seen normally at birth. Such a curve is shown in Fig. 3 (1), page 23. If this infant had not been made to sit until it had developed sufficiently to acquire the pysiological curve (2), it would not at this age show any spinal curvature. It had, however, through improper treatment reacquired the posterior curvature (1) of the early hours of life.

FIG. 40.



Posterior spinal curvature from sitting too soon.

As the child grows older, weak undeveloped muscles have a tendency to allow lateral and posterior curvatures to be produced. Habit, of course, has much to do with these faulty positions of later childhood.

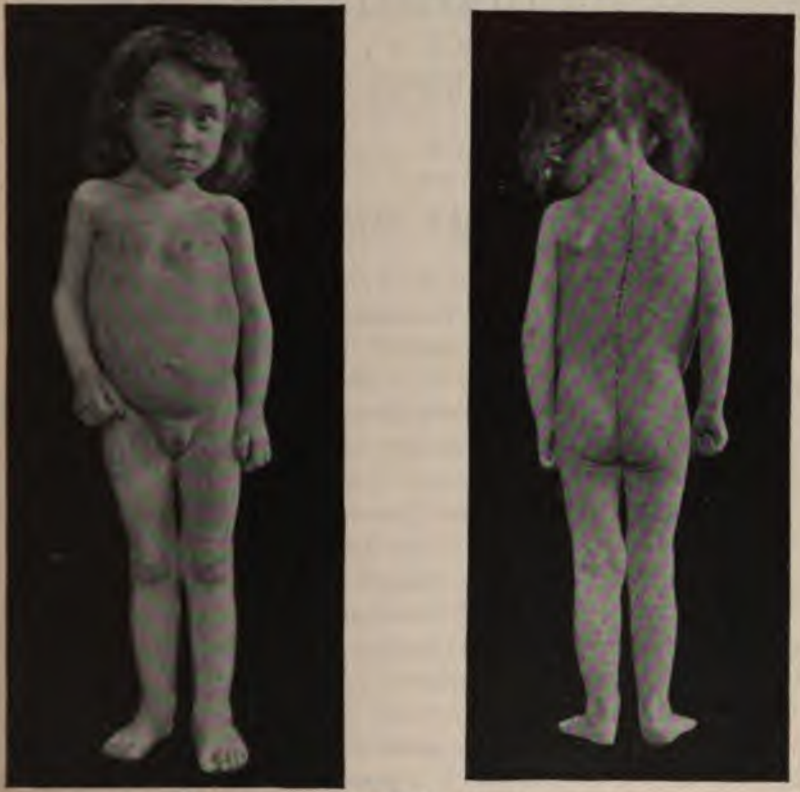
Figs. 41 and 42 represent a girl aged four and one-half years, with a lateral curvature, not from disease of the spine, but one which is usually explained as a result of superincumbent weight coming upon muscles which are unable to support it properly.

On looking at her from behind, the curve which the line of the spinal column takes to the right in the dorsal region is quite distinct. On looking at this same child in front, the right shoulder is seen to be higher than the left, and the whole thorax is in a distorted position. These deformities are always more readily recognized by looking at the child in front and preferably across the room, as the outline of the chest and hips is much more clearly defined on the anterior aspect of the body than on the posterior. Posteriorly, in cases even of the slightest lateral curvature, one notices the difference in the level of the angles of the scapulæ. This child stooped, and had what is commonly called round shoulders.

In any case of round shoulders lateral curvature should be thought of and carefully eliminated.

Faulty attitudes in sitting and standing play a great rôle in producing these curvatures. We must, however, acknowledge that such spinal curvatures have been differently explained on the ground that they are the result of a lack of development of all the tissues upon one side of the spine. Other explanations have also been given; but in certain individual cases it is impossible to formulate any reasonable cause for the curvature.

FIG. 41. FIG. 42.



Lateral curvature of the spine. Child 4½ years old.

LEGS.—At birth the infant's legs are curved rather than straight, as is seen in Figs. 28 and 29, facing page 86. The natural tendency of the growth of the legs is to become straight, but if the child is encouraged to stand and walk too soon, especially if the bones have not been properly nourished, the weight of the head and trunk becomes too great to be supported by the legs, which curve outward in the form of an ellipse, a condition which is called "bow-legs." The deformity of "knock-knee," in which the leg at the knee bends in rather than bows out, may occur from simple weakness, but is rare except in rhachitis, under which it will be more fully described.

DIVISION II.

FEEDING.

GENERAL PRINCIPLES.

Just as the highest aim of medical art should be directed to the province of preventive medicine, so the highest and most practical branch of preventive medicine should consist of the study of the best means for starting young human beings in life. They should be preserved from the perils which surround the early hours of their existence, and be given strength and vigor to resist the attacks which must inevitably be made on their vitality, and which are greater and more dangerous in inverse proportion to their age. With these objects in view, the preventive medicine of early life becomes pre-eminently the intelligent management of the nutriment which enables young human beings to breathe and grow and live. In fact, it is a proper or an improper nutriment which makes or mars the perfection of the coming generations. The feeding of infants is, then, the subject of all others which should interest and incite to research all who are working in the domain of pediatrics. The subject is a great one, and is worthy of the most careful study. The responsibility of discussing so serious a question is a grave one. It should be taken up carefully. It should be dealt with broadly. We must acknowledge that in the status of feeding, as it has existed up to the last few years, the average human breast-fed infant was more likely to live, other conditions being the same, than the infant which was fed by any other method. But we must remember that the latest investigations of this subject show very clearly that it is not human milk as a whole which is pre-eminently good, but that it is a varied combination of the different elements of the milk which makes it the best food during the first year of life. It is our province to study and make use of these elements of the food, which were once somewhat mysterious, but which are now rapidly becoming known through the work of patient and careful investigators.

In reviewing the immense amount of literature which has accumulated on the subject of feeding, we find that the superiority of human milk to all other kinds of infant food in the early months of life is acknowledged so generally that it has become an axiom. On the other hand, the opinions expressed regarding artificial feeding in the past are so diverse and so opposed to one another that it is evident that much which has in former years been taught must be unlearned, or rather admitted to be untrue, before we can expect to make any decided progress in this most difficult subject.

In our endeavor to copy nature we may hope that, as our knowledge increases, more and more light will be thrown upon those points which are now obscured by ignorance. It is, indeed, of the first importance that we should recognize our ignorance, and, watching every advance which science is making in this subject, be ready to sweep aside preconceived ideas which do not rest upon established facts, and thus by wise iconoclasm build our knowledge on a surer basis.

The great number of artificial foods used by physicians according to the fashion of the day only proves that artificial feeding has never arrived at that state of perfection where it could compete with human breast feeding. The difficulty in approaching the study of the subject has been that physicians as a class have regarded it too purely from a clinical stand-point. We know, for instance, how easily we may be misled by the apparently good effects of a medicament when perhaps on further investigation, or in the light of some new discovery, we learn that the improvement in the case was due not to the drug, but rather to circumstances entirely apart from it. The same rule applies equally well to the case of many foods and methods of feeding. To state concisely what I have already referred to, we should, in studying the form of nutriment which shall be suitable for an especial period of life, manifestly be guided by what nature has taught us throughout many ages. The researches of science at present, especially in the subject of infant feeding, are wisely directed towards learning to read the truths which nature presents to us. Great progress has been made in reading these truths. What we are also endeavoring to do is to copy them, and in regard to human milk a great advance has been made in our knowledge as to what we are to copy from it.

The feeding problem is one which is surrounded with many difficulties on account of the great diversity of individual circumstances and idiosyncrasies. Certain infants thrive on peculiar mixtures which are not adapted to infants as a class. Many will not thrive on that food which nature has provided for them, and the well-being of an infant will depend much upon the circumstances by which it is surrounded, such as affluence or poverty, country or city life. The constituents of the nutriment which nature has provided for the offspring of all mammals in the early period of their existence is essentially animal and never vegetable. Human beings in the first twelve months of life are carnivora. It is therefore evident that an animal food, entirely and freshly derived from animal and not vegetable sources, has been proved to be the nutriment on which the greatest number of human beings live and the least number die.

MAMMARY GLAND.—In regard to the early months of life, a knowledge of the changes which take place in the mammary gland from many causes is of vital importance and must be kept in view. The methods of modifying the milk in the mammary gland, however limited in their scope, should be carefully investigated and adapted to the individual infant according to its age and size and general physical condition. The mammary gland, in its perfect state, uninfluenced by disease or nervous disturbance, or by the improper living of the mother, is a beautifully adapted piece of mechanism constructed for the elaboration and secretion of an animal food. When in equilibrium it represents the highest type of a living machine adapted for a special purpose,—mechanically, physiologically, and economically. When from any cause this sensitive machinery is thrown out of equilibrium, its product is at once changed, sometimes slightly, but again to such an extent that the most disastrous consequences may follow when it is taken by the young consumer. The breasts of all mammals are elaborators and producers. They are not storehouses for preserving sustenance until it is needed. They are delicately constructed mills, turning out, when demand is made for it, a product which has been directly formed within their walls from material which has been brought through their portals from various parts of the economy. The breast is a compound racemose gland, lined with glandular epithelium, which forms sugar, fats, and proteids, and these are mixed with water and salts from the blood. The epithelial cells are so finely organized, and so sensitive with their minute nerve connections, that changes of atmosphere, changes in food, the emotions, fatigue, sickness, the catamenia, pregnancy, and many other influences, throw their mechanism out of equilibrium most readily, and change essentially the proportions of their finished product. Then again this delicate mechanism adapts itself to the quantity of its product, elaborating a smaller or a greater supply, according to the demand actually made upon it by the consumer. The same breast will either supply the proper amount of milk demanded for the requirements of the average age or a greater amount for the same age in case of a greater gastric capacity. Again, this machinery is regulated as to the time which it takes to produce the average food required for the different ages, a shorter interval of feeding being needed for the younger infant and a longer one for the older. This fact is made evident by the decided qualitative changes which result when the gland is called upon to produce its product at improper intervals. Thus, a prolonged interval lessens the solid constituents in their proportion to the water, while a shortened interval, by exciting the epithelial cells to frequent work, over-stimulates them, with the result of increasing the solids in their proportion to the water. In fact, too long intervals produce a product too dilute, while too short intervals produce a product too concentrated. The analyses of large numbers of specimens of human milk at different periods of lactation show us that not only do the constituents vary from month to month, and even from day to day, but that this variation takes place as much in the early as in the later periods of lactation. We are not warranted, therefore, in assuming that the milk grows stronger as its age increases, provided that it still remains in normal equilibrium. The mammary gland acts both as a secretory and as an excretory organ, so that it cannot be classed as a metabolic tissue in the limited meaning which we now attach to these words. Yet the metabolic phenomena giving rise to the secretion of milk are so marked, so distinct, and have so many analogies with the metabolism which we meet in adipose tissue, that we must look upon the mamma chiefly as a secretory organ (Foster). This, however, is only within certain limits, for we know that at times foreign elements may be excreted from the gland. This at once suggests the interesting question as to when the mammary gland is most likely to have what we might call its normal secretory function interfered with and to assume temporarily the function of an excretory organ. This seems to occur both before the gland has attained its equipoise, as during the colostrum period, and later when any of the above-mentioned influences occur which affect the general mechanism of the gland. In these instances we find the colostrum reappearing in the milk. Therefore in the beginning of lactation, during lactation when normal metabolism is interfered with, and as lactation draws to a close, we have analogous conditions in which the mammary gland instead of being a normal secretory organ becomes abnormal and more or less an excretory organ. During these periods of abnormal gland excretion we must remember that drugs can be eliminated by the milk more freely than when the gland is in equipoise. We assume, therefore, that the mamma during that early period of lactation, which essentially represents a condition of lack of equipoise, has a double function, partly secretory, partly excretory. The greater the excretory function of the gland is at any time in proportion to the secretory, the more abnormal will be the finished product; while the nearer the gland approaches to a purely secretory organ, the more perfect and normal will be its product. The mechanism of the mammary gland is therefore in its most perfect condition after the colostrum period has ceased, and at a time when the general organism, both physical and mental, is freed from causes detrimental to a perfect metabolism.

General principles are vital in their importance when we come to study the subject of feeding in all its phases, whether the nutriment to be provided for the infant is to come directly from its mother, a wet-nurse, or an animal, or indirectly from the product of the mammary gland. These principles are, (1) That nature throughout all ages has clearly indicated by means of natural selection what the source of supply should be; that is, that the mother should during some early period of its life supply food for her offspring from her mammary glands. (2) That when, owing to disease, over-civilization, or any causes which prevent the offspring from receiving its sustenance directly from the maternal mammæ, some nutriment must be substituted which will correspond as closely as possible to the natural food-supply. (3) That this substitution can be obtained most exactly through the product of the mammary gland of another woman. (4) That, owing to the strong analogy between human beings and all animals which suckle their young, we should in our efforts to copy good human milk make use not only of what we have learned from human beings, but also of what is known of lactation as it occurs in animals. This requires a knowledge of the investigations and experience of those who have studied commercially the breeding of animals and their food, and the production and modification of their milk.

FIRST NUTRITIVE PERIOD.

The nutrition of young human beings may be divided into three distinct nutritive periods, corresponding to the degree of their development. The first period consists of the first ten or twelve months of life. The second period comprises the second and third years, and the third period the remaining years of childhood. The science of feeding depends almost exclusively, in addition to the general principles already referred to, on the knowledge of what elements of the food are required by the growing tissues in these nutritive periods, and also on the time when the various digestive functions are ready and able to dispose of them. The first nutritive period is essentially the only one in which human milk need be considered. There is a marked analogy between the nutrition of human beings and other mammals, and it is therefore necessary to understand the lactation of animals when we endeavor to explain that of human beings. I here wish to acknowledge my indebtedness to Mr. G. E. Gordon for placing at my disposal his practical observations on the feeding, breeding, and lactation of cows.

The first nutritive period represents the first twelve months of life, and is obviously the most important one of the three. In this period the infant may be fed by a number of methods. It may be nursed by its mother, or a wet-nurse, or an animal, or it may be nourished by food especially prepared from the milk of one of these.

I. MATERNAL FEEDING.

The first of these methods, the *maternal*, is so far superior to any other which has ever been known that I shall assume that it is the best, and the one from which in almost every particular all others should be copied.

Normal Maternal Conditions.—The assumption that the maternal is, when normal, the ideal source of infant food-supply presupposes many important conditions concerning the mother and the function of her mammary glands. She should be strong and healthy, of an even, happy temperament, desirous of nursing her infant, and have time to devote herself to this special duty during the whole period of her lactation. She should have a sufficient supply of milk, and should be willing to regulate her diet, her exercise and her sleep according to the rules which will best fit her for her task. These may be said to be the ideal conditions which we endeavor to obtain for an infant which is to be nursed under the most favorable circumstances. It is true that women who are far from vigorous nurse their infants with seemingly good results, and that a frail, delicate-looking mother may have an abundant supply of good milk. These are exceptions, however, which make the principles just stated all the more true. We must have some general principles to guide us in our endeavor to perfect the nutriment of infants as a class, or we shall surely in many instances do serious harm to the individual.

Contra-Indications to Maternal Feeding.—With few exceptions, the mothers who have uncontrollable temperaments, who are unhappy, who are unwilling to nurse their infants, who are hurried in the details of their life, who are irregular in their periods of rest and in their diet and exercise, are unfit to act as the source of food-supply for their infants. Even if their milk happens to be sufficient in quantity, it will probably be so changeable in quality as to be a source of discomfort and even of danger rather than the best nutriment for their offspring. It is far better for such mothers not to attempt to nurse, but to adopt some other method of feeding. It is of still greater importance that mothers who are suffering from some chronic disease, or one which their infants may directly inherit, should give up all thoughts of nursing. When there is no question of disease in the mother, it is our duty to investigate, and, if possible, to counteract the other contra-indications to nursing, often only caused through ignorance of what to us seem very simple truths, but which to the young mother are enveloped in mystery. There is, then, a double necessity for studying in the closest detail the conditions which constitute normal lactation; first, that, knowing what is normal, we should recognize what is abnormal, and, by the intelligent use of our knowledge, render possible an apparently unsuccessful attempt to nurse; secondly, that we may know exactly on what the normal and vital conditions of successful nursing depend, in order that we may understand what we should copy in substitute feeding.

Nursing.—The natural method of feeding is by sucking. The infant should be placed in a comfortable position in its mother's arms, with its head and back supported. It should be made at once to understand that it is to begin its meal as soon as the breast is offered to it, and continue, with, of course, breathing-spells, until the meal is finished. The mother should herself preferably be sitting, as she can thus best manage and control the infant if it is inclined to be restless.

The formation of the lips and buccal cavity are adapted to the mechanism of suction. The breast is so organized that it provides a fresh supply of food at the required intervals. It prevents fermentation of the food before it enters the infant's mouth, while at the same time the suction incites to action both the necessary digestive fluids of the infant and the function of the gland itself. The gland avoids a vacuum by collapsing as it is gradually emptied, and allows the food to flow continuously, thus obviating the tendency to exhaustion of the infant and prolongation of the nursing-time which necessarily accompanies a retarded flow of the milk. Finally, the breast is practically self-regulated as to the amount which it is required to provide according to the infant's age. A healthy infant should empty the breast with easy and uninterrupted sucking in about fifteen to twenty minutes.

NIPPLES.—In certain cases the mother's nipple is so small or depressed that it is a source of much annoyance to the infant, and at times this interferes so seriously with its obtaining the proper food-supply that its nutrition suffers, and some other method than nursing has to be substituted. It is here that the ingenuity of the physician is taxed to its utmost. Every kind of device may fail, and it is necessary patiently to try one after the other before deciding to give up the nursing. Nipple-shields should be experimented with, and will sometimes obviate the difficulty. We should, however, always impress upon the mother the fact that the value of her milk as a food may be entirely destroyed if foreign elements are allowed to enter with it into her infant's mouth. This simply means extreme cleanliness of the glass shield and rubber nipple.

When the nipples are very tender and cause great discomfort to the mother during the nursing, their condition frequently becomes so serious an obstacle as to prevent nursing altogether. This change, however, should not be thought of for at least several days, or until it is absolutely certain that the exquisite pain is more than the mother is willing or able to endure. It is often the case that after a little time of the greatest suffering from tender or excoriated nipples the whole difficulty will pass away and the mother be able to nurse her infant with comfort. Where the nipples show a tendency to be dry and hard, it is well to apply some simple ointment once or twice a day during the last few weeks of the pregnancy. Astringents, as a rule, should not be used. Bathing with cold water before and after the nursing, and thus keeping the tissues in a healthy condition, appears to be as successful as the application of any medicaments.

MASTITIS.—Another trouble which may arise during the nursing period is a disturbance of the mammary gland itself, sometimes amounting merely to a stasis in its milk production, but again going on to inflammation. The latter is a serious matter, and should at once be placed in the hands of a skilful surgeon. The former condition requires great care in its management. Gentle massage from the periphery of the gland towards the nipple, amounting in fact to merely a delicate stroking with the ends of the fingers, is an important part of the treatment. The breast should be withheld from the infant for about twenty-four hours, and the milk from time to time drawn in small quantities by means of a properly adjusted breast-pump. The breast should also be carefully supported by a swathe. If these measures are begun as soon as there are any indications of disturbance in the breast, these abnormal conditions soon disappear. The indications referred to consist in the appearance of hard swellings in place of the usual soft elastic condition of the milk glands. These swellings may occur without any especial pain, but on palpation they are usually tender to a greater or less degree.

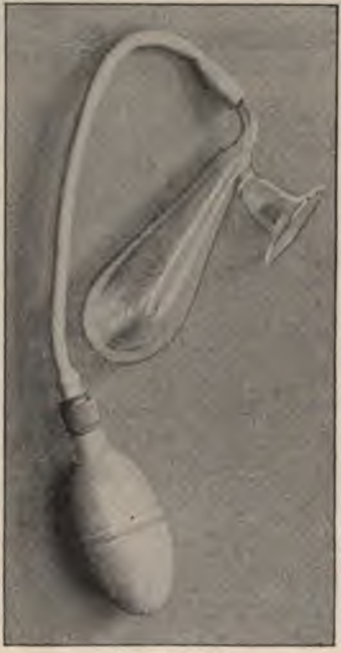
In regard to the relation of micrococci to inflammation of the breast, according to Zweifel and Döderlein there are in mastitis two varieties of organisms, the staphylococcus pyogenes aureus and the streptococcus pyogenes, but never the staphylococcus pyogenes albus. They admit that other varieties may perhaps be found on closer investigation, but at the same time they consider it striking that in all their cases there were never any local or general symptoms caused by the staphylococcus pyogenes albus, although that they were virulent was proved by their inoculation of mice. There is not much doubt that these pathogenic organisms gain access to the gland through the nipple. The infant may not be able to hold the nipple with sufficient firmness on account of some abnormal condition of the nipple itself. Under certain circumstances, even when the nipple is well formed, the infant has insufficient suction-power to obtain its food, though the food itself may be perfectly adapted to its digestion. In these cases we often find that it cannot or will not be induced to obtain its food through a shield and rubber nipple, as shown in the following case:

An infant, seven months old, was dying of starvation, as I had not been able to prepare for it a food which it could digest and thrive on. (This was before milk laboratories were established.) It was totally unable to nurse, although the breast-milk was a good one and agreed with it perfectly when it was introduced into its mouth with a spoon. The milk was pumped from the breasts at regular intervals and given to it from a bottle for over three months with the greatest success, the infant thriving, and at the end of that time being in a perfectly healthy condition.

Breast-Pump.—In regard to the use of the breast-pump there is a great difference of opinion, but I believe that those who have opposed its use have been influenced to a great degree by what they have seen in their hospital practice, and also by the views of others who have, in like manner, met with unfortunate results in lying-in hospitals. It is well known that all inflammatory conditions about the breast are more likely to occur in hospitals than under conditions in which the woman is less likely to be exposed to pathogenic organisms. This should be taken into account when we are deciding whether or not to use a breast-pump. In my experience, acquired in a great degree from my private practice, where every precaution in regard to cleanliness and asepsis could be obtained, I have never met with any bad results from the use of the pump. Fig. 43 represents the form of pump which I have found safe and useful.

The apparatus should be one which can be carefully cleansed, and should, therefore, preferably be made of glass. No one special pump will, in all probability, suit every case, and it is of importance that one should use the greatest care in adapting the pump to the individual. When applied to the breast it should cause little or no pain or discomfort. The part which is adapted to the nipple is like an ordinary nipple-shield. This is attached to a glass bulb, into which the milk falls as it is drawn from the breast. The mechanism is very simple. A vacuum can be produced in the glass bulb by means of suction through a rubber tube attached to a rubber bulb with its valve working backward.

FIG. 43.



Breast-pump.

MILK.—The products of the mammary glands of all mammals is essentially the same. In all we find substances representing the great subdivisions of food-stuffs,—that is, fats, carbohydrates, proteids, and salts,—but there is considerable variation in the relative proportions in which they occur in the milk of different mammals, and, as regards their ultimate chemical analysis, much is still to be investigated by the analytical and physiological chemists. It is primarily on the results of their work that the advances in the scientific feeding of infants and children are to be made. In general the composition of milk consists of *fats*, of *carbohydrates* in the form of lactose or milk-sugar, of *proteids* in the form of *caseinogen* (casein) and *lactalbumin*, of *salts*, *extractives*, and *water*. It is the combination of these various elements which makes the resulting product characteristic of the especial mammal.

FORMATION.—The more recent investigations on the physiological action of the mammary glands tend to show that the actually dissolved constituents of milk do not have their source in a simple process of filtration or diffusion from the blood, but are dependent upon a specific secretory activity of the glandular elements (Hammarsten). Evidence of this lies in the fact that milk-sugar is not found in the blood, that the lactalbumin is not identical with serumalbumin, and that the mineral bodies exist in milk in different proportions from those in the blood-serum of the same animal. According to Foster, milk is the product of the activity of certain protoplasmic cells, occurring in the epithelium of the mammary gland. These cells are rich in proteids and nucleoproteids, which seem to be the source of the casein or its mother-substance. The protoplasm itself becomes a constituent of the secretion (Hammarsten).

When the milk is kept at 35° C. (95° F.) outside of the body, the caseinogen is increased at the expense of the lactalbumin. When, in cows, the action of the cell is imperfect, as at the beginning and end of lactation, the lactalbumin is in excess of the caseinogen; but when the cell possesses its proper activity, the formation of caseinogen is in the greater proportion.

The fat of milk is produced within the protoplasm of the epithelial cells, and is set free by the destruction of the fat globules; a portion of the fat is probably taken up by the glands themselves from the blood and eliminated with its secretion. That this is possible has been proved by the experiments of Winternitz, in which the passage of iodized fats in the milk was noted; and similar observations were made by Spampani and Daddi with sesame oil. Microscopically, the fat can be seen to be gathered in the epithelial cell in the same way as in a fat cell of the adipose tissue, and to be discharged into the channels of the gland either by a breaking up of the cells or by a contractile extension very similar to that which takes place when an amœba ejects its digested food. A formation of fats from carbohydrates in the animal organism is now positively proved, and it is possible that the milk glands themselves also produce fats from the carbohydrates brought to them by the blood (Hammarsten). It is also well established that a part of the fat of milk is produced from proteid material; but the question in all these instances as to how much fat is produced by the secretory mechanism of the milk glands, and how much is obtained from other organs and tissues and eliminated from the blood by the milk glands, has not been determined.

The origin of the milk-sugar is not known. That it is formed in the cell protoplasm is indicated by the fact that it is not found in the blood itself, and may be maintained in abundance in the milk of carnivora which are fed exclusively on lean meat. One of the nucleoproteids mentioned above as occurring in the cell protoplasm yields a reducing substance when boiled with dilute acids, and future investigations may show some relation between this substance and lactose.

NERVOUS DISTURBANCES AFFECTING THE MILK.—The secretion and elimination of milk are very evidently under the control of the nervous system, which produces marked changes in both the quantity and the quality of the mammary product in proportion to the relative nervous excitability of the special mammal. Women are especially sensitive in this respect, and when living in the midst of our modern civilization, so harmful for the production of good nursing, present an exaggerated example of disturbance of the equipoise of the mammary gland. The chemistry of the equipoise and lack of equipoise of the mammary product appears to be closely connected with its proteid element. This element is known to be a compound one and decidedly complex, but for purposes of illustration we can safely say that the word proteid is a general term, which includes caseinogen and lactalbumin; also that these factors of the complete whole vary in their proportions to each other according as the mammary function is or is not in a state of equipoise. In the colostrum period, and probably in the analogous periods represented by the abnormal conditions already spoken of, the lactalbumin is in excess in proportion to the caseinogen, while as the equipoise of the function becomes more complete the caseinogen is increased proportionately to the lactalbumin. Probably at the end of lactation, as in the beginning, we shall find this same condition of richness of lactalbumin and deficiency of caseinogen. This increase of the the lactalbumin at the expense of the caseinogen explains how the excretory function of the gland at times becomes more prominent than the secretory.

These nervous disturbances, however, may also cause an over-production of the total proteids, as shown by their percentages. In some cases also the fat has been found to be much reduced in its total percentage. Instances of this have arisen when, as observed by Zukowski, seasons of fasting with their accompanying excitement of the emotions have induced such a disturbance of the equilibrium of the milk that the fat has been found to be decreased to the low percentage of 0.88, with the result that the infant has become sick and given evidence of impaired nutrition. These same nervous influences in all probability have to a greater or less degree their analogy in the milk-product of all mammals.

COLOSTRUM.—During the early days of lactation the mammary gland secretes a fluid which differs somewhat from that which is produced later. The milk at this period is called *colostrum*, and the period is called the *colostrum period*, on account of the presence in the milk of certain elements called *colostrum corpuscles*. These cells measure from 12 to 22 μ in diameter, and show a small, irregular, extensively degenerated nucleus. Their protoplasm contains large and small granules, which show the proteid reactions, and are not stained by acid, basic, or neutral dyes; a few of the granules which stain by osmic acid are probably fatty. The corpuscles have been described by Czerny as lymphoid cells, whose function is to absorb and reconstruct unused milk globules and to convey them from the milk glands into the lymph-channels. They disappear from the mother's milk in a week or ten days after birth. If they continue in the milk into the third week, or return at any time during lactation, they almost invariably cause disturbance of the infant's digestion, and become an indication for a temporary suspension of nursing. If they persist for any length of time, the mother's milk is manifestly not suitable for the infant, and a substitute should be provided.

CHEMISTRY.—An analysis of colostrum milk of a cow by Harrington gave the following results:

Fat	1.71
Milk-sugar	4.90
Proteids	1.72
Ash	0.79
Total solids	9.12
Water	90.88
	100.00

The table which follows represents the analyses of five specimens of human colostrum milk, also made by Harrington:

TABLE 24.

	I.	II.	III.	IV.	V.
Fat	1.40	0.68	2.40	5.73	4.40
Milk-sugar and proteids	9.44	11.53	11.15	10.69	11.27
Ash	0.17	0.31	0.25	0.16	0.21
Total solids	11.01	12.52	13.80	16.58	15.88
Water	88.99	87.48	86.20	83.42	84.12
	100.00	100.00	100.00	100.00	100.00

The most recent study of human colostrum has been made by Woodward, who analyzed the colostrum of six nursing women, using in each instance the combined twenty-four hours' amount of the middle milk, and following each case from three to seven days. He concludes as a result of his investigations that colostrum corpuscles are not always found in the so-called colostrum-milk; that when they are present the percentage of proteids is higher, and as they disappear the proteid percentage drops. In all his cases there was a loss of weight in the infants varying from eight to twelve ounces, whether the colostrum corpuscles were present or absent; when they were absent a high temperature in the mother was noted. The results of his analyses may be best seen in the following table:

TABLE 25.

		General Average of Twenty-six Analyses.
Color	Yellowish.	....
Reaction	Alkaline.	....
Specific gravity	1024 to 1034.	1029.5
Fats	2.0 to 5.3 per cent.	4.0 per cent.
Proteids	1.64 to 2.22 per cent.	1.9 per cent.
Ash	0.14 to 0.42 per cent.	0.2 per cent.
Total solids	10.18 to 13.65 per cent.	12.5 per cent.
Lactose (calculated)	5.6 to 7.4 per cent.	6.5 per cent.
Water		87.5 per cent.

The observations of Townsend have shown that the infants of multiparæ do not lose as much weight in the colostrum period as those of primiparæ, and also that the shorter the colostrum period the smaller the so-called physiological loss of weight in the new-born.

Colostrum is also supposed to have a laxative effect, and so to aid in removing the meconium. Whether this action is of any advantage to the infant is doubtful. The appearance of the colostrum corpuscles is simply an indication that the equilibrium of the mammary gland has not been established, or has been disturbed. It may be that the not infrequent disturbance of the infant's digestion in the early weeks of nursing is produced by an exaggerated abnormal condition occurring in the colostrum period, as well as by the return of the colostrum at irregular intervals.

HUMAN MILK.—Beyond the general conditions affecting the mammary products of the mother, which have already been spoken of, I know of no way of increasing the flow of milk. I have little confidence in galactagogues in the form of drugs or special foods, for their number betrays their inefficiency. The milk becomes lessened from many causes, some of which are identical with those which commonly produce any disturbance of its equilibrium such as have been mentioned. Certain drugs, such as belladonna, will in some women cause a notable decrease in the flow of milk, and must therefore be given with care during the nursing period. An active cathartic, and also a diet composed of solid food and a small amount of water, will also lessen the quantity of milk.

CLINICAL EXAMINATION OF HUMAN MILK.—The quality of the mother's milk is of so much importance to the welfare of the infant that it is of the greatest practical assistance in the management of certain cases to know the results of an analysis of the breast-milk. This can be done exactly only by an analytical chemist. The methods of analysis used is too purely a chemical question to be of practical use in clinical work, but simple approximate clinical tests are often very desirable, even though they are less accurate, when the services of a skilled chemist cannot be obtained.

To obtain a specimen for analysis the hands should be sterile, the breast and nipple should be carefully washed with sterilized water, and from 20 to 30 c.c. (5 to 8 drachms) of milk drawn by the breast-pump, which, being made of glass, can also be thoroughly washed. The milk should then be poured into a sterilized bottle, tightly corked, and kept on ice until the examination is made.

The general methods of making the rough quantitative estimation of the constituents of the milk are as follows:

Specific Gravity.—This is obtained by means of the hydrometer, for the use of which only 15 c.c. (½ ounce) of milk are required.

Fats.—In every case it is very important to know the exact percentage of the fat, both from its being the most variable element and from its use in the determination of the percentages of the other elements. The most exact means for this purpose outside of the chemical laboratory is an apparatus called the *Babcock Fat-Tester*, which is shown in Fig. 48, and described on page 192. As this is not an expensive machine, it has seemed to me that in communities at a distance from an expert chemist, or where the people are unwilling to pay for a complete analysis, a Babcock machine could be owned jointly by a number of physicians and kept at some central place. The smallest amount of milk required for determining the percentage of fat with the Babcock machine is 17.50 c.c.

Another less accurate method of estimating the percentage of cream is as follows:

A glass-stoppered cylinder is filled with milk exactly to the upper line, which is marked 0. A pipette should be used for putting the last few drops into the cylinder, care being taken not to allow the milk to run down the inner side of the tube, since this somewhat obscures an exact reading. The cylinder is then corked and allowed to stand for twenty-four hours at a temperature as near to 21.1° C. (70° F.) as is practicable. A variation of a few degrees on either side of this point is unimportant. If, however, the variations are wide, the rapidity with which the cream rises is somewhat modified.

In the great majority of cases the lower line of the cream has become sharply defined at the end of twenty-four hours, and can then be recorded. If this is not the case, the milk should be allowed to stand for six hours longer before reading the percentage.

By comparing the percentage of the cream with that of the fat, as determined by a chemical analysis of the same specimen, it has been discovered that the ratio of the cream to the fat is very nearly 5 to 3, and for clinical purposes it can be so estimated.

Proteids.—There is no known method of determining directly the percentage of the proteids in the milk by a clinical examination, and a complete chemical analysis by an expert is the only one that can be accepted as accurate. It is possible, however, from a knowledge of the specific gravity and the percentage of the fat, to make an approximate calculation in regard to the percentage of the proteids, at any rate sufficiently close to determine whether in a given case they are near the normal, or are in very large or very small proportions. Holt has described a method in which the only instruments needed are a small hydrometer, a pipette, and a glass-stoppered cylinder graduated to one hundred parts and holding about 10 c.c.

The specimen of milk for analysis should be taken from the "middle milk," and it is important that the milk should be freshly pumped and handled as little as possible, also that the graduated glass cylinder should be scrupulously clean, otherwise the milk will often sour before the cream has had time to rise. This is particularly true in summer. 15 c.c. ($\frac{1}{2}$ ounce) is the amount of milk required for the test.

In estimating the proteids certain suppositions must and can be fairly accepted:

(1) Supposing the proteids to remain unaltered: if the percentage of fat be low, the specific gravity will be high, but if high, the specific gravity will be low.

(2) Supposing the fat to remain unaltered: if the percentage of the proteids be high, the specific gravity will be high, but if the percentage of the proteids be low, the specific gravity will be low.

If, therefore, the fat and the specific gravity be known, the proteids may be estimated by the following rules:

(1) If the percentage of the fat be found to be high and the specific gravity high, that is, from 1033 to 1034, we may assume that the proteids are also of high percentage, otherwise the excessive fat would bring the specific gravity below the normal average.

(2) If the fat be found to be of low percentage and the specific gravity high, we may assume the proteids to be nearly normal, since the high specific gravity is explained by the small proportion of fat.

(3) If the percentage of fat be high and the specific gravity low, the proteids may be assumed to be normal, since the variation in the specific gravity is explained by the high percentage of fat.

(4) If the percentage of fat be low and the specific gravity low, the percentage of the proteids is also low, since otherwise the small proportion of fat would make the specific gravity above the average.

Of course it is only the wide variations in the proteids which can be recognized by these rules; but these variations are often very important.

We can then say that, knowing the specific gravity, and calculating the fats as three-fifths of the known percentage of the cream, we can judge whether the proteids are normal, very high, or very low in amount. Whereas only wide variations can be recognized by these rules, even these are often very useful. Holt asserts the estimation of the proteids of milk by this method to be as accurate as the estimation of solids by the specific gravity in examinations of the urine.

Another method of estimating the percentage of proteids has been recommended by Woodward. Two "milk-burettes," each of a capacity of 10 c.c., having a glass pinch-cock or valve and a narrow exit-tube about one inch long, each containing 5 c.c. of milk, are subjected to a temperature warm enough rapidly to sour the milk—35° to 37.8° C. (95° to 100° F.). They are allowed to remain in this temperature until a distinct precipitate can be seen, which is in from eighteen to twenty-four hours. At the end of this time the milk has distinctly separated into an upper layer of viscid yellow fat, and a lower layer of fluid milk, quite opaque above, transparent below, and clinging to the sides of the tube. At the bottom there is a granular precipitate. The burettes are then cooled in water, the milk serum withdrawn into two graduated tubes, and 10 c.c. of Esbach's solution (picric acid, 5 gm.; citric acid, 10 gm.; water, 500 c.c.) is added up to the 15 c.c. mark. The mixture is then stirred with a glass rod and placed in a hand centrifuge. The amount of centrifugation required is in direct proportion to the care used in separating the fat, and should be continued until there is a constant reading. This can be quickly accomplished if the fermentation of the milk in the tubes is watched and the separation of the casein is made as soon as the precipitation is formed. The reading expresses in percentage the amount of total proteids in the milk.

Sugars and Salts.—These may for clinical purposes be assumed to be about constant in all human milk, and in fact are practically shown to be by a large number of exact chemical analyses.

MICROSCOPIC EXAMINATION.—The mere microscopic examination of milk beyond the determination of the presence or absence of colostrum corpuscles and foreign matters, such as pus, blood, and epithelial cells, is too uncertain and misleading to be in any way depended upon, the chemical analysis being the only practical method which can be recommended. The truth of this statement was lately impressed upon me when a physician skilled in the use of the microscope sent me a specimen of woman's milk which he stated was rich in fat, but which the analysis showed to have only a little over one and a half per cent. of this element.

The presence of an undue amount of yellow coloring matter is at times very misleading. I have also seen human milk which had a greenish color, evidently produced by some of the micro-organisms which are known to occur in cow's milk, but the nature of which is not yet fully determined and which under the microscope are not represented by anything abnormal.

CLINICAL SIGNIFICANCE OF THE CHEMISTRY OF HUMAN MILK.—There is no doubt of the great value of an expert chemical examination of the milk in cases where an infant is not thriving, although apparently receiving a sufficient quantity of milk from its mother. On the other hand, we must remember that a chemical analysis will never give any information regarding the quantity of the milk, and it often happens that when such an analysis has proved the quality to be good, the infant is not thriving because the quantity of the milk is very small. The symptoms which indicate that it is the quantity of milk which is at fault rather than the quality are that the breasts at the nursing time are soft, and that only a small quantity of milk can be extracted from them by the breast-pump. A period of nursing longer than the usual fifteen to twenty minutes before the child is satisfied should make us suspicious that the milk is lacking in quantity. We can also determine the actual quantity of the milk which the child has imbibed at an especial nursing by means of weighing, as described on page 59. A number of observations at different nursings in the day must, however, be made before a correct conclusion can be reached by this latter procedure.

König, Forster, Meigs, Harrington, and others have analyzed the milk of a large number of women of all nationalities. An average of their results is expressed in the following table:

TABLE 26.

Average Human Milk.

Reaction	Amphoteric or slightly alkaline.		
Specific gravity	1028 to 1034.		
Water	87	to 88	per cent.
Total solids	12	to 13	per cent.
Fats	3	to 4	per cent.
Milk-sugar	6	to 7	per cent.
Proteids	1	to 2	per cent.
Total mineral matter	0.1	to 0.2	per cent.

Reaction.—According to Hammarsten, woman's milk is amphoteric in reaction instead of alkaline. Courant has shown by delicate chemical tests that the relation between the alkalinity and acidity in breast milk is as 3 to 1.

Specific Gravity.—The specific gravity varies normally to a considerable degree on account of the variations in temperature to which the milk happens to be exposed at the time when the specific gravity is taken. When, however, the milk has its average normal composition, and the temperature to which it is exposed is 15.5° C. (60° F.), its average specific gravity is 1031.

Water.—One of the most important chemical facts to be remembered for clinical purposes is the very large proportion of water which is found in normal human milk, for it shows it to be a highly diluted food by which the best results can be obtained in infant feeding. It also indicates the care that should be taken not to overtax the comparatively slight power of absorbing a concentrated food which exists in the early months of life.

Fat.—The fat of human milk is made up chiefly of palmatin, stearin, and olein. About two per cent. of the total fat consists of the glycerides of butyric, caproic, caprylic, and myristic acids. The production of animal heat is so very important a part of the infant's well-being that it is not surprising we should find so large a percentage of fat as well as of sugar in the food which is provided for it. The presence of fat in the milk is not only for the purpose of nutrition, but also as a means for the maintenance of bodily heat. This latter function of the fat cannot with impunity be trifled with, and is essential for active metabolism. A proper amount of fat is probably of great aid in the regulation of the fæcal discharges. An amount of fat proportionate to the proteids is presumably necessary, or at least of great aid, in their proper digestion. We should naturally expect that unless the standard percentage of fat, or at least a near approach to it, existed in the mother's milk, trouble would be likely to arise with her infant, and this corresponds with my experience in cases in which the special ingredient which has interfered with the success of the nursing has been the fat. I have found clinically that when the fat was much lessened the nutrition suffered, that the digestion was not good, and that there was a tendency to constipation, while when its percentage was decidedly above the standard the digestion was weakened, there was a tendency to diarrhœa, and in consequence a resulting poor nutrition.

These clinical observations at once suggest that in the management of infant feeding we must recognize the existence of two important conditions. One of these is the digestion of the infant, the other is its nutrition. These two requirements for a successful lactation are based on the facts that the milk may be easily digested but not nutritious, and that it may be highly nutritious but difficult to digest, so that it is the equilibrium of these two conditions which produces a perfect infantile development. It is especially important that the percentage of fat in an infant's food should be within the limits of the normal variations which are found in the milk of healthy nursing women with healthy infants. For, although it is admitted that a large percentage of surplus fat is frequently found in the fæces of infants whose digestion and nutrition are normal, and whose food is human milk, yet we have no more right to conclude from this that a small percentage of fat is sufficient for nutrition, or that a large surplus will be eliminated by the fæces, than we have to assume that there is too much oxygen in the blood because we find a certain surplus of oxygen in the arterial blood which is returned to the lungs in the pulmonary veins. In fact, it is far more probable that nature introduces a certain percentage of fat into human milk with a purpose which can be accomplished only by that percentage, so that it is an error to change this percentage beyond the variation which commonly occurs in average human milk.

Sugar.—The form of sugar which is found in human milk is called milk-sugar, or lactose, and has the highest percentage of all the elements constituting the total solids of the milk. The sugar is more digestible than the fat, but does not have so much potential energy—that is, so much heat-producing power in a given weight—as does the fat, which is to the sugar as 2.4 to 1. The conversion of milk-sugar into lactic acid gives rise to many of the changes occurring in milk.

Proteids.—Although there have been a great many different opinions expressed as to the average percentage of the total proteids in human milk, we are led at present to believe that it is normally one or two per cent. Proteid is a general name including caseinogen and lactalbumin, which in its general features resembles ordinary serum-albumin, but the ultimate chemistry of these elements is still obscure. We recognize that this lactalbumin is present in small and variable quantities when the mammary gland and its secretion are in a normal condition, while at the time when the glandular function is being established, and during periods of glandular disturbance, it becomes proportionately larger in amount. According to König, who, in his comparative analysis of the caseinogen and lactalbumin in human milk, estimates the total proteids at 1.82, the caseinogen has a percentage of 0.59, while the lactalbumin is 1.23. The proteids, as a whole, are a valuable source of information to us when we are determining whether the milk is normal or abnormal, and in arranging an infant's food the relative proportions of the caseinogen and lactalbumin should be borne in mind.

Mineral Matter.—The mineral matter, which is sometimes called the ash and sometimes the salts, has an average percentage of from 0.1 to 0.2. Up to the present time, although a certain number of analyses of the mineral matter of human milk have been made, yet the results, for various reasons, have been deemed unsatisfactory. So large a quantity of milk is needed for a reliable determination of the percentage of each element which makes up the total amount, that this in itself has been an important reason for failure in accuracy. The determination of the mineral matter of cow's milk has not been attended with the same difficulty, and its percentages have been estimated with comparatively reliable results. It has always been supposed that there is a radical difference between the percentages of the mineral matter of cow's milk and that of human milk. The exact knowledge of the percentages which exist in the latter has become of still greater importance since such decided advances have been made in the modification of the elements of the former. With the view of making some advance in this difficult question, and of providing for the milk-modifiers of the future a more exact basis for perfecting a substitute food resembling as closely as possible the product of the human breast, I undertook, in the spring of 1893, to procure an unusual and sufficient quantity of human milk for analytical purposes. In the course of a few weeks, by means of the concerted action of numerous assistants, I collected five and a half liters (about six quarts) of human milk, which is an unusually large quantity for experimental purposes. This milk was immediately reduced to its mineral constituents in the laboratory of Dr. Charles Harrington. The analysis of this large amount of mineral matter was then made by Dr. Harrington and Dr. L. P. Kinnicutt, with the following results:

The Mineral Matter of Human Milk.

Unconsumed carbon	0.71
Chlorine	20.11
Sulphur	2.19
Phosphoric acid	10.73
Silica	0.70
Carbonic acid	7.97
Iron oxide and alumina	0.40
Lime	15.69
Magnesia	1.92
Potassium	24.77
Sodium	9.19
Oxygen (calculated)	6.16
	100.54

Composition of the Mineral Matter calculated from the above Analysis.

Uncombined carbon	0.71
Calcium phosphate	25.35
Calcium silicate	1.35
Calcium sulphite	2.11
Calcium oxide	1.72
Magnesium oxide	1.91
Potassium carbonate	24.93
Potassium sulphite	8.04
Potassium chloride	12.80
Sodium chloride	23.13
Iron oxide and alumina	0.40
	102.45

A closer approximation to the relative proportions of the salts in the form in which they occur in milk, calculated from the above analysis, may be stated as follows:

Calcium phosphate	23.87
Calcium silicate	1.27
Calcium sulphate	2.25
Calcium carbonate	2.85
Magnesium carbonate	3.77
Potassium carbonate	23.47
Potassium sulphate	8.33
Potassium chloride	12.05
Sodium chloride	21.77
Iron oxide and alumina	0.37
	100.00

In comparing the previous analyses which have been made, and which can be found in König's Nahrungsmittel, II., 2^e Auflage, with this new analysis, we must remember that the previous analyses were made some years ago. In the last few years the processes which have been employed have been so much more exact that these results must be considered far more trustworthy than those made at an earlier date. It is not remarkable, therefore, that distinct differences should be found between this new analysis and the analyses which have hitherto been made, and presumably this last analysis is the correct one. It has been made with the greatest care, and by means of the most improved technique, by two eminently competent and well-known chemists, who in their work have acted as controls on each other. In this way great precision has been attained.

The residue obtained from the evaporation of about six quarts of woman's milk was extracted with naphtha to remove the fat, and then ignited at a very low temperature so as to prevent the volatilization of the chlorides. The ignition was accomplished by placing the residue from the naphtha extraction in a platinum dish which was supported on a platinum coil inside of a larger platinum dish, the latter being heated with a free flame. Even at this low temperature a partial change in the composition of the ash took place, the sulphates being reduced to sulphites, but not to sulphides, as the ash on being carefully tested showed that sulphides were not present. All the carbonates of calcium and all the carbonates of magnesium were reduced to oxides. The ash also contained seven-tenths of one per cent. of unconsumed carbon.

In woman's milk, naturally, there would be no free carbon. All the calcium that did not exist as phosphate would be in the form of sulphate and carbonate, not of sulphite and oxide as found in the ignited ash. The magnesium would exist as carbonate, not as oxide, and the potassium as sulphate, carbonate, or chloride. No sulphite of potassium would be present.

The chief differences between this new analysis and all previous ones are as follows:

(1) The phosphoric acid is less than half as much as previously reported.

(2) The magnesium is also less than half as much.

(3) Silica and alumina are present. They have not been returned in any previous analysis.

Assuming the truth of the statement that the constituents of the mineral elements of human milk are subject to great fluctuation according to age and other causes, it is right to assume that the mineral matter examined by Harrington and Kinnicutt, being the product of a large number of women, is a fair average specimen.

The conclusions which we can draw from these chemical analyses are far from precise, owing to the variations which may occur and to the insufficient number of reliable analyses which have so far been made, but they enable us to work more intelligently.

VARIATIONS IN MILK.—We are led to expect that we shall find that when the milk is poor and does not agree with the infant there is an excess of proteids and a diminution of fat beyond what we have so far been able to determine as the normal average percentages of these two elements. Again, when a variation takes place in the milk it is more likely to be found in the fat and proteids, as already stated, than in the sugar or the mineral matter. It is also important to have a number of analyses made, on different days and at different times, in order that the error of an especial or temporary variation may be corrected. The importance of the assistance which can be gained from these analyses is, in my opinion, very great, and many more analyses should be made than we are now in the habit of deeming necessary. The question of expense should not for a moment be considered by those who can afford to have analyses made, for not only will real benefit come to their own children through money spent in this way, but these analyses, when published and collated, will prove of great value for the proper regulation of the feeding of infants in all classes of society. An error for which we must always allow may interfere with the true analysis of the milk which the infant has actually received in its stomach at the end of the nursing, and is one which must necessarily invalidate the information which we receive from our analysis. This subject has already been referred to in speaking of the changes which arise from slight causes and influence the special specimen which is being analyzed. Thus, we should recognize that the milk varies considerably in its percentage of fat and total solids in the different periods of a nursing, and that the composition of the milk which the infant has in its stomach may differ very widely from the composition of a specimen taken directly before or after the nursing. Harrington's analyses of the three periods of a milking will illustrate the meaning of what has just been said, and although they were made from the milk of a cow, yet knowing the closely analogous conditions existing in human and in animal milk, we shall find them equally valuable in explaining the corresponding changes met with in woman's milk. They are represented in this table (Table 27):

TABLE 27.

	Fat.	Total Solids.	Water.	Mineral Matter.
"Fore-milk"	3.88	13.34	86.66	0.85
"Middle milk"	6.74	15.40	84.60	0.31
"Strippings"	8.12	17.13	82.87	0.82

Reiset and Peligot have also made analyses of the first, second, and third portions of ass's and cow's milk, with the result of showing not only the increase of solids at the end of a milking, but also that this increase is mostly of the fat, and to a lesser degree of the proteids, and that a short interval of nursing increases the solid constituents in proportion to the water, the reverse of this being true when the intervals are long. Heidenhain explains this physiological phenomenon by saying that his investigations point towards the fact that during the pauses between the milkings the cells of the glands are growing. During this time a proportionately small amount of solids and a proportionately large amount of water are secreted, while the irritation of milking causes increased activity of the milk-cells, with a corresponding increase in the solid secretion and a lessening of the water.

Harrington's analyses of woman's milk showing the "strippings" of a two-hours interval and the "fore-milk" of a twelve-hours interval are also of considerable interest:

	"Strippings," 2-hours Interval.	"Fore-Milk," 12-hours Interval.
Total solids	15.32	10.14
Water	84.68	89.86
	100.00	100.00

With these chemical and physiological facts before us, we are forced to acknowledge that we must be very circumspect in the conclusions which we deduce from such analyses of human milk as have been made up to the present time. An error in these conclusions, when a correct chemical analysis has been made, is less likely to occur from the sugar and the mineral matter than from the proteids and the fat.

Reasoning from the strong analogy which must exist between human milk and cow's milk, and being aware of the great variations which occur in the latter, we may assume that human milk is liable to vary in its composition considerably with different milkings on the same day, and also with the milkings of the same hours on different days, so that at present we are not in a position to state that our knowledge of human milk is sufficiently exact to justify an attempt to formulate a table to show the composition of woman's milk at different periods of her lactation, however valuable such information may in the future prove to be. We must also understand that human milk of normal quality, and proving to be equally-nutritious to the special infants that are fed on it, may vary considerably in the percentages of all its elements, and in the combinations of these percentages. This fact is well illustrated in the following table, showing the analyses of fourteen specimens of human milk all differing in the combinations of their different elements:

TABLE 28. (Harrington.)

Human Breast-Milk Analyses.

(*Mothers healthy, and infants all digesting well and gaining in weight.*)

	I. Per Cent.	II. Per Cent.	III. Per Cent.	IV. Per Cent.	V. Per Cent.	VI. Per Cent.	VII. Per Cent.
Fat............	5.16	4.88	4.84	4.37	4.11	3.82	3.80
Milk-sugar.....	5.68	6.20	6.10	6.30	5.90	5.70	6.15
Proteids........	4.14	3.71	4.17	3.27	3.71	1.08	3.53
Mineral matter .	0.17	0.19	0.19	0.16	0.21	0.20	0.20
Total solids. ...	15.15	14.98	15.30	14.10	13.93	10.80	13.68
Water.........	84.85	85.02	84.70	85.90	86.07	89.20	86.32
	100.00	100.00	100.00	100.00	100.00	100.00	100.00

	VIII. Per Cent.	IX. Per Cent.	X. Per Cent.	XI. Per Cent.	XII. Per Cent.	XIII. Per Cent.	XIV. Per Cent.
Fat......	3.76	3.30	3.16	2.96	2.36	2.09	2.02
Milk-sugar.....	6.95	7.30	7.20	5.78	7.10	6.70	6.55
Proteids	2.04	3.07	1.65	1.91	2.20	1.38	2.12
Mineral matter	0.14	0.12	0.21	0.12	0.16	0.15	0.15
Total solids.....	12.89	13.79	12.22	10.77	11.82	10.32	10.84
Water.........	87.11	86.21	87.78	89.23	88.18	89.68	89.16
	100.00	100.00	100.00	100.00	100.00	100.00	100.00

All these specimens of milk were obtained from healthy mothers, and in every case the infant was thriving. In a number of these cases, however, when one of the infants which was doing well on its own mother's milk was fed with one of the other combinations, it soon became sick, and had to be changed back to the one adapted to its digestion. Human milk may, then, be considered to represent not an especial food but a combination of foods, and its fat, sugar, proteids, and mineral matter to represent these different foods. In other words, we find by experience that the digestive capabilities of infants differ, just as do those of adults, and that nature provides a number of varieties of good human milk adapted to the varying idiosyncrasies of infants.

BACTERIOLOGICAL EXAMINATION.—Although human milk is usually considered to be sterile, except in some cases in which the woman is diseased, yet Cohn and Neumann have examined the milk of forty-eight healthy women and have found bacteria in forty-three cases. These organisms were mostly represented by the staphylococcus pyogenes albus, with a few of the staphylococcus pyogenes aureus and the streptococcus pyogenes. They found fewer bacteria when the breast had been emptied a short time previously, and more when there had been a stagnation of the milk in the breast. More bacteria were also found in the first few drops than in the last ones, and from their experiments they concluded that the bacteria enter the nipple from without. The conclusions deduced from their experiments, as well as from the experiments of others who have met with similar results, seem to show practically that bacteria can enter the ducts of the nipple and penetrate to a greater or less distance; also that the milk in its course from the gland to the nipple washes out the bacteria, and that we can in this way account for the presence of these organisms in the milk which is first drawn from the breast, and their absence from that which comes later.

MANAGEMENT OF FOOD IN EARLY DAYS OF LIFE.—Young animals at birth begin to receive their nourishment immediately, and a corresponding increase in their weight takes place from the first day of life. The human infant in like manner should begin with its nursing early, getting what it can from the breast until the full supply of milk has come. In this way it will not be so likely to have a large initial loss of weight to regain, a condition by which it is often handicapped at the very beginning of its career, when there is most danger to be apprehended from a depression of its vitality. Every day, every hour, is of the utmost importance in the early days of life, and provided it can be done without detriment to the condition of the mother, the sooner the infant is put to the breast the better it will be. During the first twelve hours of life, and in most cases during the first twenty-four to thirty-six hours, owing to the inability of the mother to supply milk for her infant, scarcely any food is, as a rule, obtained. If during this period the infant is restless and evidently hungry, 5 to 10 c.c. (1 to 2 drachms) of a sugar solution may be given at intervals of two or three hours. This solution should be made by dissolving milk-sugar in sterilized water, and its strength should be from five to six per cent. If the mother's milk is delayed still longer, something additional must be given to the infant, and in these cases the following prescription is useful:

PRESCRIPTION 2.

Fat	1.00
Sugar	5.00
Proteids	0.50

Reaction slightly alkaline.

10 feedings, each 30 c.c. (1 ounce). To be heated for thirty minutes at 75° C. (167° F.).

INTERVALS OF FEEDING.—The younger the infant the greater the metabolic activity, and hence the greater need of frequent feeding, for food is required not only for repair of waste, but also for the infant's rapid proportionate growth. This, with the increased demand for additional animal heat, makes essential the regulation of the intervals of feeding according to the age.

The intervals constitute a very important part of the management of breast feeding, when the quantity is regulated by the breast itself. These intervals should be definitely stated to the mother at different times throughout the nursing period, and should be adhered to. The following table represents the intervals for an average breast-fed infant, but it should be understood that the intervals of feeding should be made to correspond to the stage of development of the individual.

TABLE 29.

The day feedings are supposed to begin at 6 A.M. and to end at 10 P.M.

Age.	Intervals.	Number of Feedings in 24 hours.	Number of Night Feedings.
From birth to 4 weeks	2 hours	10	1
" 4 to 6 "	2 "	9	1
" 6 to 8 "	2½ "	8	1
" 2 to 4 months	2½ "	7	0
" 4 to 10 "	3 "	6	0
" 10 to 12 "	3 "	5	0

When the milk has begun to be produced in the breast, the infant should be fed once in two hours during the day and once during the night until it is six weeks old. The day feedings are usually reckoned from 6 A.M. to 10 P.M. This interval of two hours should be adhered to, allowing that exceptional circumstances may arise in which the physician must judge according to the individual case, until the sixth or eighth week is reached, when the intervals may be made two and one-half hours, and the number of feedings in the twenty-four hours eight. At about the fourth month the intervals can be made three hours, and the number of feedings six. When the infant is two or three months old, the night feeding can be omitted. The number of feedings at ten months may be reduced to five. Allowing the mother to have as many hours of continuous sleep at night as possible is especially important, in order that she may not be exhausted by the lack of that regular and sufficient rest which is of the utmost necessity for the production of a normal milk.

Irregularity in nursing, too frequent nursing, and too prolonged intervals often so disturb the quality of human milk as to transform a perfectly good milk into one entirely unfitted for the infant's powers of digestion. Thus, too frequent nursing lessens the water and increases the total solids in human milk, making it resemble in a certain way condensed milk; while too prolonged intervals result in such a decrease of the total solids as to render an otherwise good milk too watery and unfit for purposes of nutrition, however well it may be digested. The lesson that may be drawn from these facts is that some general rules for the feeding intervals should not only be recommended but enforced. The mother should neither injure her infant's digestion by nursing it too frequently, and thus giving it a too concentrated fluid, nor, by neglecting to feed it often enough, interfere with its nutrition by giving it a food that is too diluted.

REGIMEN OF LACTATION. **Diet.**—The diet of the nursing mother should not essentially differ from what would be considered to be a healthy one for her at any time. There is no special diet which, under all circumstances, is best for all nursing women during the period of their lactation. In the early days of the puerperium there is, as a rule, more danger of overfeeding than of underfeeding the mother. The tendency is to give too much meat and solid food, with the result that when the secretion of the milk is being established the total solids are increased to a degree beyond the capacity of the still undeveloped digestive function of the infant. Infants in the early days and weeks of life thrive better on a milk that shows a high percentage of water in proportion to that of the total solids. A rule which has in my experience become almost an axiom is that the age of the individual infant is in inverse proportion to its powers of absorbing solid food, and in direct proportion to the need of a large amount of water in its food. A light and plentiful diet should therefore be given to the mother while she is confined to her bed. This diet should consist of milk, gruels, soups, vegetables, bread and butter, and after the first week a small amount of meat once during the twenty-four hours. When the mother is able to go out of the house again, and has resumed her usual habits, the quality of the diet can be very much increased, and she can have the usual variety of food represented by meats, vegetables, milk, fruits, and cereals. There are no special kinds of food which are contra-indicated, provided we keep the food within the limits of the ordinary articles which commonly represent a plain but nutritious diet. It is very important for the nursing mother to have her meals at regular intervals, and during the early part of the lactation to take food somewhat more frequently than when she is not nursing. The additional meals, as a rule, should be made up of milk or cocoa. There does not seem to be any advantage in adding any special beverages, such as beer, malt, or stimulants, to her diet. She should receive as much milk as is compatible with her digestion, and should drink a plentiful supply before retiring at night. This wide range of food for the nursing mother has been recommended with a purpose. The food of the nursing woman is without doubt closely connected with that which she provides for her infant. Various substances are eliminated by the mammary gland, and we should therefore impress upon mothers the importance of a carefully arranged diet when they are nursing. Certain vegetables, and sometimes fish, will in individual cases affect the milk and cause discomfort to the infant. We must, then, in every case, seek to determine which article of diet may cause disturbance in the special woman's milk secretion, and eliminate that article. We should, however, be very careful not to prohibit this special article of diet from the regimen of a large number of women to whom it might be of benefit rather than of harm, simply because it has affected the milk of a few women. For the average woman a plain mixed diet, with a moderate excess of fluids and proteids over what she is normally accustomed to, will, as a rule, give the best results.

Exercise.—Exercise has so constant an influence on the changes which take place in the daily secretion of the milk, that the mother should be encouraged to be out of bed and to walk about her room as soon after her confinement as is possible without injuring her physical condition. Exercise is so important for promoting the proper elaboration and equilibrium of the milk secretion during the entire period of lactation, that it should always be insisted upon, and regular hours for walking should be as definitely arranged during the day as the hours for eating. The exercise must, however, be in accordance with the strength of the special woman, for fatigue has the same deleterious influence on the production of the milk as has lack of exercise.

DISTURBED LACTATION.—The disturbances which are liable to occur in the course of lactation are frequent and varied. They should be studied carefully and recognized at once when they occur, or the continuation of the lactation may not only be interfered with but be prevented entirely. When colostrum corpuscles are found after the first two weeks of life the milk should be looked upon with distrust, and special efforts should be made to discover the reason of their persistence, and to prevent the dangers which are liable under these circumstances to arise. These dangers may be not only from combinations of the milk elements which are incompatible with the infant's digestion, but also from the disturbances which may arise from the free mammary elimination of foreign material.

DRUGS.—We know that during periods of mammary disturbance there is a much greater possibility, than when the gland is in a normal condition, of the direct transudation from the blood of such inorganic substances as arsenic, antimony, lead, iodide of potash, mercury, and others, taken by the mother. Well-authenticated cases come to our notice from time to time in which injury has been done to the nursing infant in this way, and in which even death has occurred from the elimination by the breast-milk of certain organic substances, such as colchicum and morphine.

The greatest variety of substances have been found in the milk, but no definite rule as to the amount of this elimination has yet been established, so that our knowledge of the existence of this process is valuable as a prophylactic against harm, rather than as a means of direct benefit to the infant in disease.

We must also recognize the clinical fact that this elimination may occur at any time during the nursing period in the breasts of women who, so far as we can ascertain, are in a perfectly healthy condition. Thus, every practitioner has at times doubtless observed the laxative effect on the infant of such drugs as compound liquorice powder given to the mother; and a case has lately come to my notice where an infant vomited for weeks while taking the milk from the breast of its mother, who was unusually well and strong, but who was in the habit of drinking a considerable quantity of porter daily. After the porter was omitted the vomiting ceased at once, and did not return.

These facts warn us that the use of drugs during the period of lactation should be far more limited than at other times, and that the medicinal treatment of disease in infants is exceedingly inexact. Saline cathartics may not only act unfavorably on the infant through the mammary excretion, but may lessen very decidedly the flow of the milk, and even stop it altogether.

Menstruation.—In deciding whether a return of the menstrual period necessarily contra-indicates the continuation of nursing we cannot adopt and follow an inflexible rule, but must be guided by what seems best for the individual case. Infants are at times affected so seriously by the alteration in the constituents of the milk which occurs once in four weeks that their nutrition is markedly interfered with, and a change to a more stable food is indicated. Again, the only disturbance which may arise is a temporary and slight digestive attack for a day or two, which apparently does not materially affect the infant, and makes us hesitate to run the risk of depriving it of a food on which it thrives during twenty-six days out of twenty-eight. It is better not to be too hasty in concluding from the bad symptoms in the infant that we should at once withdraw it permanently from the breast, for the catamenia may appear once, and then not again for a number of months, the infant's powers of digestion in the mean time becoming so much more fully developed that they are unaffected by the milk of the catamenial period. Even when the catamenia recur regularly, the disturbance which may have been great at one period may for many reasons fail to recur at the next; so that the question is reduced to whether the composition of the milk shows a recovery of the equilibrium of its constituents within a few days, or remains affected to such a degree as to endanger the integrity of the infant's nutrition.

My own experience is in favor of allowing the infant to continue with the breast, unless it is decidedly contra-indicated by circumstances such as have just been mentioned. I have seldom met cases which could not without permanent injury be tided over the small amount of temporary digestive disturbance which may arise. There have, as yet, been too few analyses made during the catamenial period to justify us in drawing any definite conclusions as to the chemical status of the question; but the probability is that the milk will be found to be deficient in fat and to have its proteids increased, following the general rule of disturbed mammary secretion, and that consequently it is in a condition to interfere temporarily with both digestion and nutrition.

Pregnancy.—A much more serious question arises when the nursing mother becomes pregnant; for here the almost universal clinical experience is that the infant, for various reasons, cannot continue to be fed by its mother, it being unusual for a woman to have sufficient vitality to nourish properly her living child and growing fœtus. The danger of reflex miscarriage from the continual irritation of the mammary gland by nursing I personally have had no experience with, but this is mentioned as one of the dangers contra-indicating the continuation of nursing by a pregnant woman. We must, however, here also not judge hastily, but take all the circumstances of the case into consideration before deciding on a measure of such vital importance to both child and fœtus. If the mother remains strong and vigorous, and the analysis of her milk shows no deterioration, while the infant is a delicate one just beginning to thrive on its rightful supply of natural food, or if it is during a hot period of the year, and especially when a wet-nurse or feeding from a milk-laboratory cannot be employed, it will often be wiser to take some risk and continue the nursing for a certain time, perhaps six or eight weeks, and then, according to circumstances, gradually to substitute another food. Almost every case will differ in the questions to be decided, and must be judged on its own indications and contra-indications, always, however, recognizing the accepted rule that lactation and pregnancy are usually incompatible.

Management of Disturbed Lactation.—The nursing mother is inclined to believe that if she feels well and strong her milk must be good for her infant under all circumstances. She therefore frequently transgresses the rules which are necessary for keeping her milk in equilibrium, and she should be made to understand that sometimes abnormal variations are liable to arise, however good her general health may be. She is simply fulfilling a task demanded by nature from those who bear children, and her duty, when once she has undertaken to nurse, is to prevent as much as possible these variations by regulating her life to a normal standard and avoiding excitement. Both of these requisites of a normal lactation come within the province of the physician to explain as he would any other branch of rational medicine. He should impress upon her that emotional mothers do not make good nurses, and that the physiological influence of the emotions on the nervous system, with its resulting changes in the mammary secretion, has necessarily a much wider range in women who are subjected to the customs and vicissitudes of modern life than it has in those who live in a more natural way.

The following table shows the percentages and combinations which are likely to occur in abnormal milk:

TABLE 30.

Showing typical analyses of a normal, a poor, an over-rich, and a bad human breast-milk.

	Normal Milk. (Healthy life as to exercise and food.)	Poor Milk. (Starvation.)	Over-rich Milk. (Rich feeding; lack of exercise.)	Bad Milk. (Pregnancy, Disease, etc.)
Fat	4	1.10	5.10	0.80
Sugar	7	4.00	7.50	5.00
Proteids	1.50	2.50	3.50	4.50
Mineral matter	0.15	0.09	0.20	0.09
Total solids	12.65	7.69	16.30	10.39
Water	87.35	92.31	83.70	89.61
	100.00	100.00	100.00	100.00

The terms poor and bad milk are merely relative, and in common use do not have a definite meaning. I have adopted the terms for the purpose of simplicity and to distinguish a milk which can be restored easily to a normal condition from one in which the difficulty of such restoration is very great. By a poor milk I mean one which represents a condition of lack of nourishment or starvation in the mother, but one which can easily be changed by the proper feeding of the mother. In this case the normal mechanism of the mammary gland has not been interfered with. By a bad milk I mean one which represents a profound disturbance of the mechanism of the mammary gland produced by many causes, disease, pregnancy, and especially extreme nervous conditions in the mother, and one which cannot be easily changed to a good milk.

The following are some of the rules which will be found of use in managing a case of disturbed lactation:

General Principles for Guidance in managing a Disturbed Lactation.

To increase the total quantity	Increase proprotionately the liquids in the mother's diet, and encourage her to believe that she will be enabled to nurse her infant.
To decrease the total quantity (Rarely necessary.)	Decrease proportionately the liquids in the mother's diet.
To increase the total solids	Shorten the nursing intervals; decrease the exercise; decrease the proportion of liquids in the mother's diet.
To decrease the total solids	Prolong the nursing intervals; increase the exercise; increase the proportion of liquids in the mother's diet.
To increase the fat	Increase the proportion of meat in the diet and of fats which are in a readily digestible and assimilable form.
To decrease the fat	Decrease the proportion of meat in the diet.
To increase the proteids (Very rarely indicated.)	Decrease the exercise.
To decrease the proteids	Increase the exercise up to the limit of fatigue for the individual.

In attempting to formulate rules we must understand that we are dealing with a subject of which very little is known definitely. I can, therefore, at present only state my experience in a large number of cases, and give some general idea of how we can recognize whether we are dealing with a bad or poor milk rather than with a normal variation of a good milk. This knowledge of the variations which take place in human milk is of the utmost clinical importance during the period of lactation, for it is the only means by which we can decide definitely and intelligently many vital questions in this period.

Instances have continually been brought to my notice where infants have been allowed either to continue with their mother's milk when they were not thriving on it, simply because it was mother's milk, or, on the other hand, have been weaned from their mothers for what would evidently have been insufficient reasons had the case been thoroughly understood. In both instances a proper knowledge of what can be done with human milk—that is, with the management of its different constituents by increasing or decreasing their relative proportions—would have been of benefit to both mother and child, and in some cases would have saved the life of the latter. This lack of knowledge, or rather lack of adaptation of the knowledge which we possess of this branch of medicine is, to say the least, reprehensible, and in other branches of our art, which are more intelligently and carefully studied, would be deemed inexcusable. Physicians are continually stating to their patients that human breast-milk is the best food for infants, and at the same time are content to ignore the very principles which would make their statements true. We should understand that when we speak of the superiority of breast-milk as a food, we mean good average breast-milk and for the average infant.

In all these cases of disturbed lactation we must determine whether the symptoms in the infant are really caused by a disturbance of the milk-supply. We ascertain first whether the supply of milk is sufficient in quantity by the methods already described. We then investigate the quality of the milk. A chemical analysis shows us whether the percentages of the different elements are (1) normal or (2) abnormal. If we find them to be normal, we know that it is not the milk which is disturbing the infant, and we must seek for the cause of the disturbance in other sources beyond the breast. If we find the percentages to differ decidedly from those of average human milk, we must determine whether it is the variation from the normal average percentages or combinations of percentages which is producing the trouble, or whether these percentages are really well adapted to the infant and the cause of the trouble is to be looked for elsewhere. This can be done only by changing the different percentages and their combinations and watching the result. If we find them abnormal, we can usually determine whether it is one or several of the elements which are producing unfavorable symptoms, and we should endeavor by our treatment to change the percentages of these elements so as to correspond first to the normal average percentages, and then, if this is not sufficient, to reduce them to lower percentages than the average until the infant's digestive functions have recovered their equilibrium. We must not forget in applying these principles that the cause of the disturbance of the milk may exist in some abnormal condition of the mother, whether physiological or pathological, and that this cause must first be removed or we shall fail to regulate the milk.

A sedentary life, with abundance of rich, mixed food, provided the woman has a strong, healthy digestion, appears to increase the total solids and to decrease the water. This increase is almost always in the fats and proteids rather than in the sugar and mineral matter; in fact, the marked variations in human milk are almost always shown in the fat and proteids, and hence our attention must almost invariably be directed to correcting these elements. This is fortunate, as we know of no special treatment, except on very general principles, by which we can alter the proportion of sugar or salts to the other constituents. A meat, or rather a nitrogenous, diet and fat in an easily digestible and assimilable form increase the fat in breast-milk. The proteids are more difficult to deal with. They have a tendency to increase in very bad and in very rich milk. The problem which we have to solve is almost always how to decrease them, no matter what the milk is. Our knowledge, unfortunately, concerning a sure means of reducing the proteids is very limited. Practically, however, I have found that when the woman is in good health it is physical exercise which we must insist upon, preferably walking in the open air and within the limits of fatigue. A walk of from one to two miles twice daily I have found to be about what the average healthy woman in New England needs to reduce the percentage of the proteids in her milk; but the amount of exercise must be carefully regulated according to the physical capabilities of the individual.

Bearing in mind these simple rules, and having determined, by means of an analysis or analyses, the cause of the special disturbance, it is often possible to regulate the nursing period in cases in which a lack of this knowledge would necessitate weaning. In this way also serious harm to the infant may be avoided.

The following tables represent the changes which took place in the milk in certain cases of disturbed lactation.

TABLE 31.

(*Human Milk.*)

Showing the influence of a luxurious life on a poorly fed but healthy wet-nurse.

	I. Normal.	II. Two days before change of food.	III. Rich food and but little exercise for a month.	IV. Food and exercise regulated.
Fat	4.00	0.72	5.44	5.50
Sugar	7.00	6.75	6.25	6.60
Proteids	1.50	2.53	4.61	2.90
Mineral matter	0.15	0.22	0.20	0.14
Total solids	12.65	10.22	16.50	15.14
Water	87.35	89.78	83.50	84.86
	100.00	100.00	100.00	100.00

TABLE 32

(*Human Milk*)

Showing a bad milk and one which it was impossible to manage on account of the continued recurrence of the same cause, uncontrolled emotions.

	Normal.	Emotions causing disturbance in infant's digestion.
Fat	4.00	0.62
Sugar	7.00	5.80
Proteids	1.50	4.21
Mineral matter	0.15	0.20
Total solids	12.65	10.83
Water	87.85	89.17
	100.00	100.00

TABLE 33.

(*Human Milk.*)

Showing a milk possible to manage, because the mother, though excitable, was able and willing to control her emotions.

	Normal.	Infant doing badly. Colic. Mother before treatment.	Infant doing well. Mother after treatment.	Wet-nurse provided but not used
Fat	4.00	1 62	3.20	3.04
Sugar	7.00	6.10	6.40	6.60
Proteids	1.50	3.54	2.52	2.32
Mineral matter	0.15	0.17	0.18	0.12
Total solids	12.65	11.43	12.30	12.08
Water	87.35	88.57	87.70	87.92
	100.00	100.00	100.00	100.00

In the above case the mother was very nervous and wished to nurse her infant, but thought that she could not, as she had been discouraged by her nurse and physician. She was then told that she could nurse in a week, if in the mean time she took proper food and exercise and withdrew the infant from the breast. This she did, and had her breasts regularly pumped, with good results.

TABLE 34.

(*Human Milk.*)

Showing the effect of the catamenia on human milk.

	Normal.	Catamenia, Second Day.	Seven Days after Catamenia.	Forty Days after Catamenia.
Fat	4.00	1.37	2.02	2.74
Sugar	7.00	6.10	6.55	6.35
Proteids	1.50	2.78	2.12	0.98
Mineral matter	0.15	0.15	0.15	0.14
Total solids	12.65	10.40	10.84	10.21
Water	87.35	89.60	89.16	89.79
	100.00	100.00	100.00	100.00

TABLE 35.

(*Human Milk.*)

Showing a milk in which the proteids, which were disturbing the infant, could not be reduced until the mother was made to walk comfortably, and thus without fatigue.

	Normal.	Infant with colic and vomiting. Mother taking no exercise and very rich food.	Infant as before. Mother walking two miles daily, but having blisters from uncomfortable shoes.	Infant doing well. Mother walking two miles. Easy shoes, no blisters.
Fat	4.00	3.05	0.65	3.34
Sugar	7.00	6.10	5.25	6.80
Proteids	1.50	3.89	3.82	2.61
Mineral matter	0.15	0.16	0.18	0.16
Total solids	12.65	13.20	9.90	12.41
Water	87.35	86.80	90.10	87.59
	100.00	100.00	100.00	100.00

TABLE 36.

(*Human Milk.*)

Showing how a milk can be managed while the nursing is continued.

	Normal.	Infant two weeks old, with serious general nervous symptoms and pain. Mother eating much meat and taking no exercise.	Mother walking and eating less meat. Infant entirely well.	Infant four months old, with pain and diarrhœa. Mother not walking so much.	Infant doing well. Mother walking two miles daily. Milk diluted one-fifth.
Fat..........	4.00	3.44	2.09	3.98	3.19
Sugar........	7.00	5.60	6.70	7.00	5.60
Proteids......	1.50	3.96	1.38	2.22	1.78
Mineral matter	0.15	0.20	0.15	0.19	0.16
Total solids...	12.65	13.20	10.32	13.89	10.73
Water.......	87.35	86.80	89.68	86.61	89.27
	100.00	100.00	100.00	100.00	100.00

As is seen from the last analyses, the infant did not do well until the mother began to exercise, and at four months it was again affected by apparently the high percentage of the proteids. The infant was considerably under the weight corresponding to that of the average infant of four months. It was found to nurse twenty-five minutes at a time, and by calculation from its weight before and after nursing, it was found to take from 80 to 120 cc. (20 to 30 drachms). This amount being larger than the probable size of its stomach demanded, the time of the nursing was reduced to twenty minutes, and 20 c.c. (5 drachms) of sterilized water were given in the middle of the nursing, thus changing the percentages in the milk to the figures which are represented in the last column. This calculation is on the basis of 100 c.c. (25 drachms) to each nursing.

So long as this method of feeding was adhered to, the infant did well. It was evidently a case in which the infant could not digest over two per cent. of proteids.

TABLE 37.

(*Human Milk.*)

Showing that even for a long interval the breasts may be pumped and the result be a successful nursing.

	Normal.	Infant showing nervous symptoms and much uric acid. Mother taking no exercise and much rich food.	Infant showing no uric acid and thriving. Mother walking two miles and not eating much meat.
Fat	4.00	5.71	2.67
Sugar......................	7.00	4.00	6.60
Proteids	1.50	4.29	3.18
Mineral matter............	0.15	0.19	0.17
Total solids................	12.65	14.19	12.62
Water	87.35	85.81	87.38
	100.00	100.00	100.00

In this case the infant was withdrawn from the breast temporarily, and the breasts pumped for twenty-seven days. When the analysis presented the figures seen in the last column, the milk was treated by diluting it, as in the previous case, and the infant was put back to the breast.

TABLE 38.

(*Human Milk.*)

Showing the value of retaining the breast-milk by managing even an unpromising case.

	Normal.	Infant with colic and failing. Mother no exercise, nursing irregularly, irregular and improper sweet food. Nervous, worried condition.	Infant put on bottle. Breasts pumped every four hours. Moderate exercise,—one mile. Full regular diet. Tranquil.	Exercise increased to two miles. Small amount of meat.	Eating much meat. Exercise the same.
Fat	4.00	0.34	3.24	2.79	4.84
Sugar	7.00	5.40	5.45	5.05	6.00
Proteids	1.50	3.61	3.95	3.66	3.42
Mineral matter...	0.15	0.18	0.16	0.20	0.17
Total solids	12.65	9.53	12.80	11.70	14.43
Water...........	87.35	90.47	87.20	88.30	85.57
	100.00	100.00	100.00	100.00	100.00

The last case represents a bad milk from the failure of the healthy mother to conform to the rules of lactation. This bad milk, represented in the second column, had to be made into a rich milk by regular feeding before any attempt could be made to alter the ratio of the constituents. The proteids were then reduced somewhat by exercise, and, after the breasts had been pumped for two weeks, the analysis showed the percentages as represented in the last column. The milk was then diluted with sterilized water, and the infant was put to the breast and did well; in fact, was carried through an attack of retro-pharyngeal abscess with this breast-milk.

The decrease in the total quantity of the milk is of ordinary occurrence at any time during lactation, but it is most common among civilized races at about the eighth to the tenth month. When it occurs early in the lactation it is very disheartening to the mother if she is desirous of continuing her nursing. She becomes fearful that the flow of milk may stop altogether, and the nervous influence thus brought to bear on the mammary gland tends to increase the disturbance. We should therefore encourage her to believe that the milk will return. The following case illustrates what has just been said:

The mother was much discouraged because her milk lessened in quantity early in the lactation, and she was convinced that it would not return. She had been taking, without my knowledge, a disproportionately small amount of fluid in her diet. There was an element in this case which the intelligent nurse brought to my notice,—namely, that the infant was not vigorous, and when put to the breast sucked feebly and called upon the gland for very little milk. Reacting to this lack of stimulus, the gland, although in a normal condition, secreted only the small amount demanded by the infant, and the milk lessened day by day. Treatment was instituted on the supposition that the mammary gland is practically self-regulating as to the amount of food which it will elaborate at a given nursing. If it happens to be called upon to nourish twins, it will increase the amount of its supply. If the infant which is put to it has a small gastric capacity, it will produce the amount needed for that capacity. I assured the mother that the milk would return, and I treated directly the mammary gland itself. An increase was made in the amount of liquid in the mother's diet, and the breasts were, after each nursing, pumped gently, skilfully, and thoroughly. The breast-pump supplemented the feeble action of the infant, and when more work was required of the gland it began to produce more milk. The increase in the liquid diet supplied the gland with materials to work with, and its mechanism ceased to be disturbed by the nervous influence emanating from the mother. She became cheerful when she found the milk returning, while the infant, now that the milk could be procured more easily, demanded more, sucked more vigorously, and thus satisfied the sensitive mechanism of the mammæ.

The next case points to the possibility of our being at times too hasty in the decision to deprive an infant of its mother's milk.

The mother, a rather delicate primipara, twenty-five years of age, was delivered of a boy seven pounds in weight. Within four hours puerperal convulsions set in, from which she recovered, but was left with albuminuria 0.25 per cent. and casts. The latter disappeared in a few days, but the albumin, although somewhat diminished, continued; and the patient, naturally of a calm disposition, was in a highly nervous condition, fearing that she could not nurse her infant, but decidedly opposed to having a wet-nurse. The milk appeared in considerable quantity on the fifth day, but the infant did not thrive, and, although it gained somewhat in weight, was very fretful, slept very little, and looked ill, so that the attending physician became alarmed, and after treating it for its dyspepsia without much success until it was five weeks old, and finding that there was still about 0.25 per cent. of albumin in the mother's urine, decided with me that the breast-milk should be withheld until we could determine the cause of the trouble, and an analysis was accordingly made, with the following result:

Fat	1.62
Sugar	6.10
Proteids	3.54
Mineral matter	0.17
Total solids	11.43
Water	88.57
	100.00

This analysis suggesting the probability that the large amount of proteids was causing the disturbance of digestion, and that the small amount of fat was not sufficient for nutrition, the attending physician was very anxious to procure a wet-nurse; but while we were endeavoring to get a proper one, we decided to empty the mother's breasts with the breast-pump every day, thus relieving her from the worry of attempting to nurse her infant and seeing it fail to gain. She also obtained in this way undisturbed nights and a great deal of out-door life. The infant was in the mean time placed on a substitute food, which was digested very well, and, as it ceased to cry, the mother's mind became tranquil, and the albumin in her urine in a few days was reduced to a trace. The treatment was carried out for a week, the milk continuing to flow freely, and an analysis (I.) was then made of the mother's milk and also of that of a healthy wet-nurse (II.) whose infant was thriving on its mother's milk.

	I. Mother.	II. Wet-Nurse.
Fat	3.20	3.04
Sugar	6.40	6.60
Proteids	2.52	2.32
Mineral matter	0.18	0.12
Total solids	12.30	12.08
Water	87.70	87.92
	100.00	100.00

The two milks being equally good, it was decided to allow the infant to begin to take one nursing daily from its mother, although the proteids were still about one per cent. higher than the infant seemed likely to digest; it was given to its mother, nursed well, seemed satisfied, digested its meal without trouble, and at six months was still being nursed and was thriving.

The next case illustrates the principle that too frequent nursing lessens the water and increases the total solids in human milk, making it resemble in a certain way condensed milk. It also illustrates what has been stated concerning the two important questions to be considered in the management of a normal lactation,—namely, that the digestion as well as the nutrition must be regarded. This case is one of the numerous instances of the same kind which have come to my notice, and also emphasizes the fact that infants are often weaned from the breast when there is not the slightest necessity for it.

The mother, a healthy primipara about twenty-two years old, had nursed her infant for six weeks, during which time the infant was fretful, suffered much from colic, and never seemed satisfied. There was, however, a continual gain in weight, although the fæcal discharges showed evidence of the food not being properly digested and were numerous and watery. By advice of the attending physician, the infant was weaned. The mother came to me for advice in regard to placing her infant on a substitute food. On inquiry I found that this infant had been nursed almost continuously night and day, with intervals usually of only one hour, and it was evident that the frequent nursings had resulted in producing a concentrated milk which the infant's gastro-enteric tract was rebelling against and was not digesting, although sufficient food was being absorbed to prevent up to this time any interference with the general nutrition. This infant, then six weeks of age, was deprived of its supply of good human milk in the middle of the summer simply because the important matter of changing the intervals had not been thought of as a means of improving the milk and relieving the pain and apparent hunger. There seems to be no doubt that if the milk in this case had been properly managed it would have agreed perfectly with the infant. I would also add in connection with this case that when the digestion is not carried on properly the nutrition must soon suffer, and it is only in the early weeks of a disturbed digestion that, as a rule, we find the nutrition to be unimpaired.

The next case is one of a multipara who was under my care at the City Hospital, and who up to the time of her entrance had been nursing her infant, which was thriving. The patient stated that her milk had always been abundant and of good color up to the time when she was separated from her infant, which was twelve hours previously, as she had to be away from home for that time. At the end of twelve hours the breast was found to be so distended that the breast-pump had to be applied. The milk was drawn with great ease, almost flowing of itself, and in considerable quantity, but it no longer resembled the milk of the previous nursings which had been at the proper intervals. On the contrary, it was clear, with very little color, the total solids were reduced to a minimum, and it no longer would have nourished the infant.

The treatment of this case was of course to pump the breasts every three hours until the infant could again be nursed.

As an illustration of the harm which may come to an infant from the percentage of fat in its mother's milk being too high, and also of the means to employ either to increase or to decrease the fat in breast-milk, this case will be of interest.

The mother was a healthy primipara. She had plenty of milk, but the infant suffered from colic and had very frequent watery dejections. She was eating a great deal of meat three times daily and not taking much exercise, so it was naturally supposed from the symptoms of the infant and the diet of the mother that an over-percentage of fat was one of the elements which were disturbing the lactation, and that a high percentage of proteids would also be found. The analysis proved this supposition to be correct:

Primipara. Healthy; eating much meat; not taking much exercise.

Fat	4.96
Sugar	6.60
Proteids	3.29
Mineral matter	0.17

It was therefore decided to reduce the meat to a minimum, which was done, and three days later an analysis gave the following figures:

Eating little meat.

Fat	1.73
Sugar	5.70
Proteids	3.74
Mineral matter	0.13

The milk was found to be lessening in quantity. The infant's dejections were less numerous and had more consistency; but it was not gaining, and continued to have pain. In fact, the analysis showed a poor milk, or even a bad one, as represented by the usual combination of a low percentage of fat and a high percentage of proteids. The woman was consequently made to eat a moderate amount of meat, and to exercise more, and three or four days later the analysis showed an improvement in the fat:

Eating moderate amount of meat; taking more exercise.

Fat	2.42
Sugar	5.50
Proteids	3.55
Mineral matter	0.15

The infant now began to gain in weight, but continued to have colic, as was expected from the high percentage of proteids. The exercise was still further increased, and a later analysis showed a decided lessening of the proteids.

Exercise still further increased.

Fat	2.35
Sugar	6.25
Proteids	2.69
Mineral matter	0.15

The infant then began to have regular movements, of good consistency, and no longer had pain; it also gained regularly in weight, and looked well and strong. The mother regulated her diet, exercise, and sleep in accordance with the requirements of her infant, and her milk again became abundant.

We shall, of course, often fail in our attempts to manage the percentage of fat in this way, but this case illustrates exactly the changes which it is usually necessary to produce in order to alter a high fat percentage. The proteids also being high, there was an over-rich milk to deal with; taking away the fat-producing element reduced the fat to a low percentage; exercise reduced the high percentage of proteids, and a combination of sufficient meat and exercise finally produced a milk which could be digested.

This next case illustrates a number of points in the management of lactation. A high percentage of the proteids was creating the disturbance in the infant, and it was their final reduction through treatment that permitted the lactation to go on.

The mother, a remarkably healthy and vigorous multipara, living in the country, had a plentiful supply of milk. Her diet consisted mostly of vegetables, and she did not take much exercise. The infant was not thriving, having had continued attacks of colic, with frequent vomiting, and it did not gain in weight. The analysis showed a bad milk, which was contrary to what we should usually expect to find in the milk of a mother who was in such perfect health as this one was.

Fat	0.52
Sugar	6.80
Proteids	2.48
Mineral matter	0.15
Total solids	9.95
Water	90.05
	100.00

The mother was instructed to eat meat and walk two miles every day. One month later, as the infant had not improved, another analysis was made, which showed that the milk was in a worse rather than a better condition.

Fat	0.45
Sugar	6.15
Proteids	2.47
Mineral matter	0.16
Total solids	9.23
Water	90.77
	100.00

It was found that the mother had eaten meat but once a day, and in small quantity; also that she had not walked much. I then insisted on her eating meat three times a day, and walking three miles. This she did for two weeks, when the infant was found to have gained slightly in weight, but to still have colic and vomiting. Another analysis showed an increase in the fat.

Fat	1,53
Sugar	6,68
Proteids	2.48
Mineral matter	0.16
Total solids	10.85
Water	89.15
	100.00

During the next two months the walking was continued, and the meat increased in quantity. The infant continued to vomit and to have colic until the mother was made to ride on horseback every day, when the pain ceased, and from that time the infant gained steadily in weight, and was well and strong during the rest of the lactation. An analysis made two and one-half months after this procedure showed that at last the proteids had been reduced to come within the limits of the infant's digestion, and that the fat, although still having a low percentage, had been increased sufficiently for the infant's nutrition. Thus a bad milk was finally changed to a good one. This infant evidently could not digest a percentage of proteids approaching 2, but fortunately could be nourished on a low percentage of fat.

Fat	2.01
Sugar	6.90
Proteids	1.54
Mineral matter	0.17
Total solids	10.62
Water	89.38
	100.00

The next case was that of a poor milk.

The infant was four months old. It was perfectly well and was digesting well, but had not gained for three weeks. The mother was producing from her breasts a sufficient quantity of milk, but the analysis showed that this milk had to be modified within the breast by a regulation of the diet of the mother:

Fat	1.29
Sugar	6.05
Proteids	2.93
Mineral matter	0.12
Total solids	10.39
Water	89.61
	100.00

She was consequently made to eat an increased amount of meat, and in the course of a few weeks the infant was thriving and gaining in weight.

The next case was that of a wet-nurse whose infant was digesting well, gaining in weight, and happened to be of about the same age as that of the infant whom she was hired to nurse. In order to see if this nurse's milk would agree with the foster-infant, the nurse and her infant were brought to the house of the foster-child, and were comfortably lodged and plentifully fed.

Twenty-four hours later both infants began to have colic and green fæ
An analysis of the milk showed a high percentage of proteids :

Fat ....
Sugar ....
Proteids ....
Mineral matter ....

Total solids ....
Water ....

The nurse was then given a lighter diet with a greater proportion of liq
made to walk one mile twice daily. By weighing the infants just before a
a nursing, it was found that they took from 90 to 120 c.c. (3 to 4 ounces) in
utes. The infants were then allowed to nurse for ten minutes. 30 c.c.
sterilized water was next given to them, and they were then allowed to n
minutes longer. In this way it was estimated that they were receiving in th
120 c.c. (4 ounces) of food in which the percentage of the proteids was unde
infants ceased to have colic, and the fæcal discharges became normal.
infant was then sent away. Two weeks later the foster-infant was thrivi
another analysis of the milk showed a sufficient reduction of proteids, the ste
was omitted.

Fat ....
Sugar ....
Proteids ....
Mineral matter ....

Total solids ....
Water ....

1

During the rest of lactation the infant digested well and gained fairly in

The following case was that of a perfectly healthy primipa
infant digested her milk well and gained in weight.

The case shows how at times an infant can thrive on what appears to
percentage of some of the solids in the milk. The analysis of her milk was

Fat ....
Sugar ....
Proteids ....
Mineral matter ....

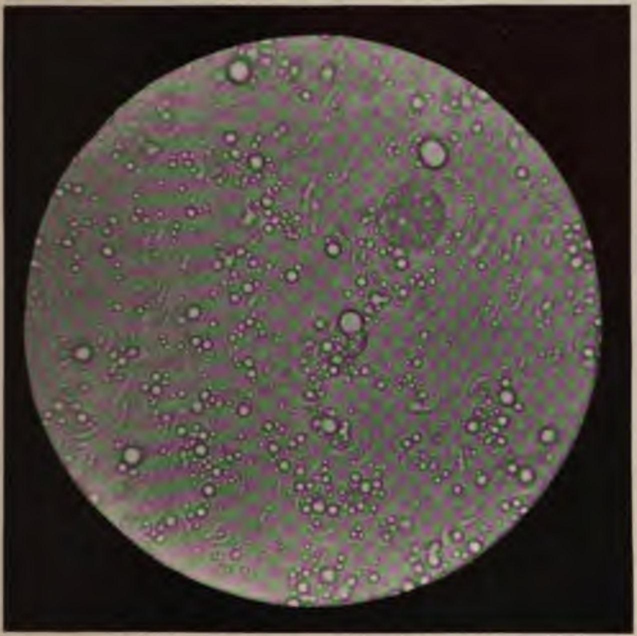
Total solids ....
Water ....

1

In contrast to this case was the following one in which the i
evidently thriving.

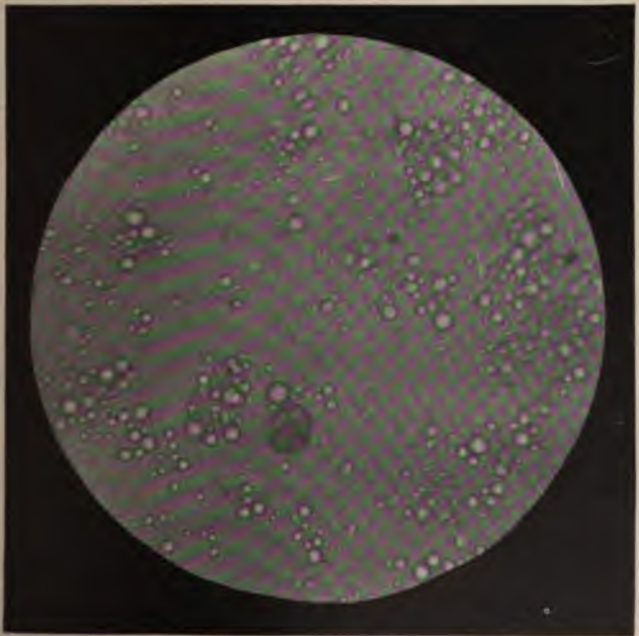
The mother was delicate and frail, and the infant was fed by a heal
wet-nurse. In the early part of the lactation the infant did not thrive,
mother was so delicate, it was not deemed advisable to attempt to improve

FIG. 44.



Colostrum milk from cow. (Photo-micrograph.)

FIG. 45.



Colostrum milk from woman. (Photo-micrograph.)

of her milk. The interesting point in connection with this case is the inability of the infant to digest a poor milk and its ability to digest perfectly well the wet-nurse's milk, which in its analysis showed a very high percentage of fats and of proteids and a low percentage of sugar:

Fat	4.72
Sugar	4.55
Proteids	4.74
Mineral matter	0.19
Total solids	14.20
Water	85.80
	100.00

In the following case it was found impossible to change the percentages of the elements in the milk.

The woman had a moderate quantity of milk, and nursed her infant for two or three months. The infant did not gain, it had colic, and at times vomited. The analysis showed that it was in the class which I have designated as "bad:"

Fat	1.61
Sugar	4.67
Proteids	4.07
Mineral matter	0.17
Total solids	10.52
Water	89.48
	100.00

An increase of meat in this mother's diet and more exercise had no effect on the percentages of the elements of her milk, and the infant was therefore weaned. Soon after beginning to take a substitute food from the milk-laboratory the infant ceased to have colic and gained in weight. The percentages of the elements in the substitute food which produced such an immediate change in the infant's condition were as represented in the prescription:

Fat	3.50
Sugar	7.00
Proteids	1.00

It was merely necessary to raise the percentages of the fat and sugar, and reduce that of the proteids, in order to produce this rapid and satisfactory result.

The next analysis is that of a woman's milk, which is instructive for a number of reasons:

Fat	2.30
Sugar	6.65
Proteids	2.57
Mineral matter	0.12
Total solids	11.64
Water	88.36
	100.00

The percentage of fat is low, and that of the proteids is rather high. The infant, with the exception of being somewhat constipated, was always well, gained in weight, and showed no digestive disturbance during the lactation. This was remarkable, as the mother's catamenia returned regularly during the lactation from the time that the infant was four months old. There was considerable flowing at the time of the catamenia, and the mother was habitually constipated and did not have a very good appetite. The infant did not seem to be affected by any of these conditions. The analysis of this milk was made from a specimen of the "middle milk," which was taken between the catamenial periods.

It may be of interest, in connection with what has been said concerning the variations in the milk which may arise from emotional causes and menstruation, to report the analysis of a milk of a mother and a wet-nurse where these influences appeared to produce certain chemical changes. The mother, a healthy but rather delicate primipara, the period of whose pregnancy had been supervised by me with the greatest care, but whose temperament was subject to extremes of despondency and excitement, was delivered, after a short and easy labor, of a healthy boy. She was exceedingly anxious to nurse her infant, but within a few hours after its birth she was seized with an uncontrollable fear that she would be unable to do so. In spite of all the assurances to the contrary which could be given to her, and the plentiful supply of milk which in due time came in the breasts, she remained in a very nervous, despondent condition. As the infant began to show decided signs of indigestion, I thought it best, before proceeding further, to investigate the composition of the milk. The analysis resulted as follows, and plainly showed the necessity of not persisting further, as it was evidently much altered from unavoidable nervous conditions, which seemed likely to recur through the whole of her lactation:

(*Mother's Milk.*)

Fat	0.62
Sugar	5.80
Proteids	4.21
Mineral matter	0.20
Total solids	10.83
Water	89.17
	100.00

Under these circumstances, a healthy wet-nurse, whose own infant was strong and thriving, was employed, and the foster-infant immediately began to gain in weight and ceased to show any digestive disturbance. After a month, however, it was found not to have made its weekly gain, to be unusually restless, and to be having frequent fæcal discharges. It was then discovered that the wet-nurse was menstruating, and on the second day the following analysis of her milk was made:

(*Wet-Nurse.*)

Fat	1.37
Sugar	6.10
Proteids	2.78
Mineral matter	0.15
Total solids	10.40
Water	89.60
	100.00

The catamenia lasted about four days, and did not return for some months. The infant after the first twenty-four hours showed no disturbance whatever, soon began to gain, and was not affected by the subsequent occurrence of the catamenia. An analysis, made one week after the catamenia had ceased, showed a decided change for the better; that is, increased fat and decreased proteids. Forty days after the catamenia a still greater improvement was found in the milk, as was anticipated from the thriving condition of the infant. The change in the percentage is shown in the following analyses :

	Seven Days after Catamenia.	Forty Days after Catamenia.
Fat	2.02	2.74
Sugar	6.55	6.35
Proteids	2.12	0.98
Mineral matter	0.15	0.14
Total solids	10.84	10.21
Water	89.16	89.79
	100.00	100.00

The following case is of considerable interest with reference to what has been said in regard to the incompatibility of pregnancy and lactation. Unfortunately, a full consideration of the condition of the milk cannot be presented, as it rapidly disappeared from the breast after the first analysis was made, and, before another specimen could be procured, had disappeared entirely.

The milk was taken from one of my patients who had been pregnant for three months and at the same time was nursing an infant nine months old.

Fat	7.64
Solids, not fat	6.04
Total solids	13.68

The infant at the breast was not thriving. It had been digesting its mother's milk perfectly and had been gaining in weight until the pregnancy had existed for some weeks. At the time the analysis was made the infant's digestion had evidently been weakened, and as a result it had ceased to thrive and was rapidly losing in weight.

This analysis will be found to illustrate several facts. In the first place, it represents a very rich food. The total solids are even greater than appear in most cow's milk, and the fat is almost double the percentage which is considered normal in both human and cow's milk.

It also shows that a food may be unusually high in the percentage of its total solids and yet not of a character suited for the nutrition of an infant. The explanation of this fact is that although for a time an infant may digest fairly well a rich food, yet that nature has provided that the percentages of the elements in its food should remain within certain limits. If these limits are transgressed, either by giving too low or too high a percentage of any of the solids in the food, the nutrition will be interfered with. In the latter case the digestive function of the infant actually becomes weakened, and the strong food soon begins to act as a foreign body. The absorption of the food is next interfered with, and the infant starves as readily on the strong food which cannot be absorbed as on the weak food in which the needed elements are lacking.

This analysis also represents a condition which, in the majority of cases of pregnancy, occurs after the first six or eight weeks,—namely, a much disturbed mammary equilibrium. The percentage of fat in proportion to that of the solids not fat is so entirely different from the percentages of the different elements in a normal milk that we may say that this milk of pregnancy represents a condition of profound disturbance.

This especial analysis must not be taken as a standard one for the milk of pregnant women, for, in all probability, analyses of milk under these conditions differ very widely, yet invariably show an absence of the normal percentages.

PROLONGED LACTATION.—In healthy women the milk towards the end of a normal lactation has a tendency to return to the condition which we notice at the very beginning of lactation; that is, the product of the mammary gland becomes unstable and the percentages show a poor or a bad milk. In rare cases I have met with women whose milk remained of fair quality and who could continue their nursing into the second year without apparent detriment to themselves or to their infants. There is, however, no reason for thus continuing the lactation, even if the mother is healthy and the milk good, for at the end of the first year, human milk, whether good or bad, is not a food which is adapted to the corresponding stage of development of the infant's digestive organs. Unmodified cow's milk and starch in some form are much better adapted to the stage of development of the digestive organs of the second year, and should therefore at that time be substituted for human milk.

MIXED FEEDING.—It not infrequently happens to nursing women, when their general health is not in a normal condition, that the supply of milk, while good in quality, is not sufficient in quantity to satisfy the infant, and the question arises whether the mother's milk should be entirely given up, or whether it should be supplemented by other food. My experience is in favor of assisting the mother to nurse her infant during the earlier months of its life. When the substitute food can be carefully regulated, and when the mother's milk is of good quality, this method is superior to that of withdrawing the mother's milk and feeding the infant exclusively upon a substitute food.

We have, on the one hand, a better opportunity for regulating the mother's milk, by increasing or diminishing the number of the substitute feedings, and, on the other hand, if the mother's milk agrees with her infant, an excellent opportunity for making our substitute food correspond to what nature has provided.

In arranging a mixed feeding we should in every case first have an analysis made of the mother's milk, and, if her milk has been agreeing with the infant, make the substitute food correspond to the maternal. It is also well to have an analysis of the mother's milk made at an early period of her lactation, as soon as the mammary gland has acquired its equilibrium and when the infant is thriving. This is a very important precaution, which may be of great use to us at a later period when the mother's milk may from many circumstances be disturbed or entirely lost. When such an accident happens, we know exactly what the composition of the milk was on which the infant was thriving, and can at once arrange a proper substitute food.

The following cases illustrate this statement:

An infant was thriving on the milk of a healthy wet-nurse. One day, without giving any warning, the nurse left the house and never returned. The infant had to be put on a substitute food, as another nurse could not be procured. It was left in the middle of the hot weather without the food which had been so well adapted to its digestion. Unfortunately, the precaution of having an analysis made of the wet-nurse's milk had not been taken, and it was some time before I was able to substitute a food which would agree with the infant.

The second case was where the mother's milk, after careful management, had become fitted for her infant, and where the infant was thriving. One day the mother received a nervous shock from seeing the arm of another of her children dislocated. Within a few hours the milk entirely disappeared from her breasts and did not return. The analysis of her milk, which had been previously made, provided me with a guide by which I could at once have a substitute food prepared which would correspond to the food which the infant had been receiving from its mother. This was done, and the infant continued to thrive, showing no bad symptoms from the change of food.

There are certain points to be considered in mixed feeding. First, if the mother's milk is agreeing with the infant, the substitute food should be of the same composition. Second, if the mother's milk is fully digested by the infant but is lacking in certain nutritive qualities, the absence of which prevents the infant's nutrition from being normal, we should, after the first week, alter the composition of the substitute food so as to make it fulfil the requirements of nutrition by increasing the percentage of that special element in the substitute which is deficient in the composition of the maternal milk.

The times at which the substitute food should be given will depend upon the number of feedings which are found to be necessary in addition to the maternal feedings, and we should carry out the same principles in this mixed feeding that have been laid down for the general management of human breast-milk. If the mother's milk is lacking in quantity we should make the intervals between her nursings longer, and introduce one or two substitute feedings according as the age of the child requires shorter or longer intervals. If, on the contrary, the mother's milk is abundant, but either too strong or too weak, we should make the intervals of her nursing correspondingly long or short. In this way, with an accurate knowledge of the percentages which exist in the mother's milk, and with our power to change these percentages in substitute feeding, we can usually in a week or ten days regulate the substitute feeding of the infant to such a degree that the mother's milk will also agree with the infant, and the infant will thrive again.

WEANING.—There is no doubt that in a considerable number of cases occurring in the practice of physicians among civilized nations the mother's milk appears to be entirely unfit for her offspring, and it becomes a question whether the infant shall be withdrawn from its mother's breast temporarily or entirely. In such an emergency the careful and repeated analysis of the milk will enable us to determine this question wisely.

I am convinced that a large number of infants are deprived of their natural food and weaned on insufficient grounds. We thus assist to keep up the resulting high mortality figures, and I believe that these figures will be sensibly reduced when, in consequence of our taking a more enlightened view of the subject, we increase the number of infants who are fed during the first three or four months of life upon a suitable breast-milk.

A particular reason among many for waiting at least three or four months before weaning is presented by the fact that the stomach, after growing rapidly, has by the fourth or fifth month become a more perfect receptacle both as to size and to function.

A number of nursing women find that at variable periods in the course of their lactation their milk begins to fail, and they are forced first to lessen the number of their nursings and then to wean entirely. The time, then, when the infant should be weaned almost always settles itself, without our intervention, at varying periods. The period of lactation, and the one which might be called physiologically normal, can, when the breast-milk remains of good quality and quantity, be carried through the first year with benefit. We have certain guides which aid us in determining the proper time for beginning to wean. Physiologically, we know that certain functions, such as that which converts starch into glucose, are but slightly developed in the early months of life, and that they are only gradually established during the first year, and not, as a rule, perfected and in a condition in which we can call upon them with impunity until the last two or three months of that year. A sign which aids us in judging the progress of this development of the functions is the appearance of the teeth, calling our attention to the fact that nature is preparing the infant to digest and assimilate a form of food different from that which it has thus far received by sucking. The presence of six or eight incisors corresponds usually in the normally developed infant to the full development of the pancreatic secretion.

A most valuable index which assures us that we need not be anxious to change the infant's food during the first year is the continuous increase in its weight, which, with a general healthy condition, results from a normal lactation. We must allow, however, for certain variations which in special cases are as important as is the rule to terminate the lactation at a definite period. The period of lactation may be curtailed or lengthened by a month or two according to the season of the year, the development of the teeth, or the condition of the child from illness or convalescence. Under such circumstances it may be wiser to feed the infant from the breast during the heated portions of the year, and to wean it in cool weather, before or after the hot season, according to the individual case. An interdental period is also preferable to a dental period, on account of the possible disturbances which may arise in the latter and interfere with the proper actions of the new functions to which reference has been made. In these exceptional circumstances, when there is any uncertainty as to the character of the milk which the infant is taking, a chemical analysis should be made at once, and repeated several times at intervals of a few days. These latter months, though not so difficult to manage intelligently as the early period of the infant's life, are much more likely to need careful supervision than the middle period, which, from its usually uninterrupted tranquillity, has been called the period of normal nutrition.

When on account of an insufficient supply of milk in the mother the infant has for some time become accustomed to several meals of a substitute food daily, the matter of weaning becomes a very simple one, for we know that we have a food which will agree with it; but when we have to begin to wean directly and to adapt a food to the infant's digestive capabilities, as in cases of sudden failure of the milk or of sickness in the mother, this procedure becomes much more intricate, and is at times fraught with considerable danger. It is in these cases that an analysis of the milk made when the mother was in good condition often proves to be of great assistance.

The method of weaning which I have adopted, and have found to be the safest and best, is the one which I have been enabled to use since having a milk-laboratory at my command. My rule is, provided that the infant is thriving or digesting its mother's milk well, to order from the laboratory a substitute food the percentages of the elements of which are very similar to what the infant has been taking from its mother. After a few days, if this food is agreeing with the infant, a change should be made in the percentages of the different elements, with the object of gradually combining these percentages in such a way as to correspond to the percentages of the elements of unmodified cow's milk. This is easily and precisely accomplished. For instance, supposing that the infant is receiving from its mother a milk in which the percentage of the fat is 4, of the sugar 6.50, and of the proteids 2, we should begin by giving the same percentage of fat (4), a lesser percentage of sugar (5.50), and an increased percentage of proteids (2.25). After a few days, if this milk is digested well by the infant, the fat can be made 4, the sugar 4.50, and the proteids 3. In a few more days, if this food is digested well, plain cow's milk, with lime-water sufficient to make it slightly alkaline, can be given. The milk which is now received from the farms connected with the laboratories is practically so free from bacteria that it need not be pasteurized in the winter months; and often, also, in the summer, it will remain fresh, except in exceptionally warm weather, or when it has to be transported a long distance. If this still agrees with the infant, cow's milk without lime-water can be given.

Unless under very exceptional circumstances, sudden weaning is to be deprecated, though of course we must admit that it is sometimes done with impunity. The safest method, so long as we cannot judge beforehand which infants will be likely to be unfavorably affected by sudden weaning, is to take plenty of time and gradually ascertain by frequent changes the food best adapted to the case. The infant should be gradually accustomed to this food, omitting the breast-feedings one by one, until finally we are sure that we have a substitute food on which it will thrive. At the tenth or eleventh month, provided that the weaning of the infant is deemed desirable at so early a period, and after having accustomed it to take plain cow's milk, starch in some form can also be given. It will be necessary to determine how much of this new element may be introduced into the infant's diet, carefully adapting the amount to its amylolytic function, which varies in different infants, and which has but lately arrived at its full development. When these changes have been accomplished, the breast can with safety be entirely withdrawn.

The danger of injudicious weaning is illustrated by the following case:

A delicate infant, backward in its development, digesting well, and a little over one year old, was suddenly deprived of the plentiful supply of breast-milk of its healthy mother and fed on oatmeal gruel. Vomiting and prostration immediately began, and continued until the oatmeal was omitted and the breast-feeding resumed, when the infant began to thrive again. Three weeks later the mother, through ignorance, suddenly and without any preparation fed it again on oatmeal gruel. On the following two days the infant vomited incessantly and was much prostrated. Several changes were then made in its food, but the symptoms grew worse, and the mother, who was now very uneasy about the infant, again put it to her breast, with, however, this time a disastrous result, as her milk from nervous influences was so changed in its quality that it acted like a poison on the infant, who fell into a condition of collapse. A wet-nurse with a healthy infant four months old was immediately procured, and after several days of complete prostration the foster-infant began to revive, and later was gradually weaned without trouble. It may be well to add, for the encouragement of physicians who have cases of this kind to deal with, that after the mother's milk had poisoned the infant, and when I first saw it, the skin was gray and cold, the fontanelle sunken, and the eyes fixed, yet recovery took place. Under the same circumstances equal success in the treatment would probably be obtained by writing for a milk prescription to contain fat 2.50, sugar 5, proteids 0.50. This, of course, would be an exceedingly weak food for an infant twelve months old, but it would be the safest combination to begin with, and could be increased in strength as the infant recovered.

II. DIRECT SUBSTITUTE FEEDING.

WOMEN.—When for any reason it is impossible or inadvisable for the mother to nurse her infant, some other food must be substituted for the maternal. The milk of another woman approaches the mother's in its characteristics most closely, and should be obtained unless contraindicated.

It is generally supposed that the mother's milk, as a rule, is more likely to be suited to her infant's digestion than the milk of another woman; but we have as yet too few cases where direct investigation by means of chemical analysis of the two kinds of milk has been made to lay down actually as a fact what we can merely grant as a supposition, that an idiosyncrasy in the mother's milk will find an analogue in her infant's digestive powers. The reverse of this proposition has also been held to be true, that at times some idiosyncrasy in the mother's milk will make it radically unfit for her infant. The probability is that analyses will show either that these varieties of milk are poor ones, or that the infants have unusually weak digestive powers.

The fact that every mother cannot provide as good a milk for her infant as can be supplied by another woman finds its analogy in the inability of some Jersey cows to rear their own calves.

The following case illustrates how at times an idiosyncrasy of digestion in the infant corresponds to some unusual percentage in its mother's milk:

The mother, a primipara, was healthy, but of a highly nervous temperament. The infant was thriving, but, as a measure of precaution in case of mammary disturbance at a later period of the lactation, an analysis was made of the milk, with the following result:

Fat	5.16
Sugar	5.68
Proteids	4.14
Mineral matter	0.17
Total solids	15.15
Water	84.85
	100.00

The report made by Dr. Harrington in connection with this analysis was, "The precipitated curd is quite similar in its appearance to that obtained in the analysis of cow's milk."

The mother was advised on general principles to take more exercise, and ten days later another analysis of the milk was made:

Fat	4.88
Sugar	6.20
Proteids	3.71
Mineral matter	0.19
Total solids	14.98
Water	85.02
	100.00

The second analysis was so similar to the previous one that, in conjunction with the perfect digestion and health of the infant, it was concluded that this infant had an idiosyncrasy of digestion which enabled it to thrive on what would in most cases cause extreme disturbance. This view of the case proved to be correct, as the infant, which was under my care for a number of months, continued to thrive. A comparison of this analysis with that of the milk of the wet-nurse on page 164, where the high percentage of proteids caused vomiting of thick curds by the infant, will show a striking similarity of the two milks. There is no doubt that in the majority of cases a milk such as is represented by these two analyses would be totally unfit, and would not only cause marked indigestion but often more serious results, such as convulsions.

The following case presents an illustration of the reverse of the supposition that the mother's milk will suit her infant's digestion better than the milk of a wet-nurse:

This infant was being nursed by its mother and showed continual disturbance of its digestion. At times it would be constipated, and again it would have attacks of colic with watery discharges. The colic was the most prominent symptom, and the child, though looking fairly well, was not gaining in weight. An analysis of the mother's milk showed that the percentage of fat was from 2 to 3, the sugar was of about the normal percentage, and the proteids varied from 3 to 3.50 per cent. The mother was of an extremely nervous temperament and was unwilling to carry out the rules for the management of her milk, which were absolutely necessary in order to reduce the high percentage of proteids, which evidently caused the disturbance. A wet-nurse was therefore procured, the analysis of whose milk was as follows :

Fat	2.96
Sugar	5.78
Proteids	1.91
Mineral matter	0.12
Total solids	10.77
Water	89.23
	100.00

The infant on taking this new milk ceased to have colic, but was more constipated and did not gain in weight. It was therefore decided that it would be wise to increase the percentage of the fat in the nurse's milk. This was done by giving her considerably more meat to eat and making her take moderate exercise. The infant within a week began to gain in weight and to sleep well, the bowels ceased to be constipated and were moved naturally every day. There was also a plentiful supply of milk. Another analysis of the milk was then made, with the following result :

Fat	3.31
Sugar	6.45
Proteids	2.36
Mineral matter	0.16
Total solids	12.28
Water	87.72
	100.00

This last analysis is of great significance. The increase in the percentage of the fat evidently regulated the fæcal movements. The total solids increased from 10.77 to 12.28, and the plentiful supply of milk made the infant gain, especially as it now was digesting perfectly. It was evident that it could digest a milk with a percentage of proteids below 2.50, while it was a percentage of 3 in the mother's milk which prevented her from carrying on her lactation.

In this case it will be seen that the milk of another woman was far preferable to that of the mother, and that the idiosyncrasy of a high percentage of proteids in the mother's milk did not find its counterpart in an idiosyncrasy in the proteid digestion of her infant.

WET-NURSES.—The general question as to whether a wet-nurse shall be employed is one which is of serious import, and must in each instance be decided by giving full weight to all of the many circumstances which are involved in the case. Foster-feeding, when all the conditions are good, is superior to substitute feeding. The reverse of this statement, however, must always be kept in view, that a poor nurse, whether from temperament, or age, or general health, or the quality of her milk, had better be set aside when the conditions are favorable for a successful substitute feeding. It is perhaps better that the nurse's milk should correspond in age somewhat nearly to that of the infant she is to suckle, but a difference of some months in age may not be a contra-indication, as we are not yet in a position to say definitely that the milk differs sufficiently in different months to make this a reason of importance in choosing a nurse. A feeble child will nurse more easily and probably have better care from a multipara than from a primipara. The preferable age of the nurse is between twenty and thirty years. Her other requisites are a condition of good health and a quiet temperament. It will save much trouble and often obviate the frequent necessity for changing if before her engagement we have made a chemical analysis of her milk; in fact, all the points which have been already referred to for a successful maternal nursing are of equal significance in the case of a wet-nurse.

The general health of the wet-nurse should be carefully investigated, as women suffering from constitutional syphilis or any chronic disease are manifestly unfit for nursing. At the same time we should be careful, unless decided symptoms of disease are present, not to set aside the milk of a delicate-looking woman until it has been analyzed. The wet-nurse in the case just described, whose milk proved to suit the infant better than did its mother's, was a frail, delicate-looking woman, but healthy. The mother, on the other hand, was a large, strong-looking woman, but of a very nervous temperament. The rapid progress which is being made in the detection of the bacillus tuberculosis, not only in the sputum but also in the milk and in other secretions, may in the future be of much practical importance in the determination as to whether a woman should nurse an infant or not, but the present state of our knowedge is only sufficiently advanced for us to state that this bacillus has been found in the secretion of the mammary gland of cows which have responded to the tuberculin test, and whose milk has been proved to be pathogenic in animal experimentation.

DIET.—The same general principles that have been stated in speaking of the diet of the mother should be applied to that of the wet-nurse. We should be extremely careful not to change suddenly the customary diet of a healthy nursing woman on purely theoretical grounds. For many years the mistake was made of keeping women on too low a diet in the early period of lactation, with the consequent delay in the establishment of a sufficiently nutritious milk-supply, and a corresponding initial loss of weight in their infants. Where, however, we are especially likely to err is in permitting a healthy, hard-working wet-nurse, accustomed to a somewhat coarse but nutritious diet, to adopt totally different habits of exercise and a diet to which she is unaccustomed, rather than to have her continue her usual mode of life. This sudden change of habits frequently results in loss of health to the nurse, with its accompanying deterioration in the quality of her milk, or at least a change in its quality so as to make it an unfit food for her foster-child. A notable instance of too radical a change of habits was brought to my notice by a case seen in consultation.

A wet-nurse had been procured for an infant ten days old. An analysis of her milk, two days before she began to nurse, is shown in the following table. Her milk was digested well for two or three weeks, during which time she was fed on an abundance of good food and rich milk. The infant then began to vomit thick curds identical in appearance and toughness with the curds of cow's milk. Another analysis was made, which showed the amount of total solids to be increased in a most marked degree, the percentage of proteids corresponding far more nearly to that of cow's milk than to that of woman's milk. The nurse was then given plainer food and skimmed milk, and the infant ceased to vomit. The infant and nurse continued well and strong during the whole year, the infant making a weekly gain in weight.

	Analysis I. Two days before change of food.	Analysis II. Rich food for a month.	Analysis III. Food regulated and milk agreeing with infant.
Fat	0.72	5.44	5.50
Sugar	6.75	6.25	6.60
Proteids	2.53	4.61	2.90
Mineral matter	0.22	0.20	0.14
Total solids	10.22	16.50	15.14
Water	89.78	83.50	84.86
	100.00	100.00	100.00

Animals.—In parts of France, notably in Brittany, infants are put directly to the cow's teats, and sometimes with good results. I know of one family of eight children, all of whom were nursed by the family cow, and all of whom grew up healthy and strong. Yet the undesirability of feeding human beings directly from the udders of animals is so manifest that this method need not be discussed.

III. INDIRECT SUBSTITUTE FEEDING.

General Considerations.—I have laid great stress upon the importance of feeding infants during the early months of life by means of human milk. We know, however, that the necessity will often arise for supplying the infant with food not from the human breast. In all probability the employment of substitute feeding will increase rather than decrease as our civilization advances. With this prospect before us, and appreciating the difficulties which in a large number of cases are liable to arise when we attempt to adapt a substitute food to the wants of an infant, it manifestly becomes a duty to endeavor to reduce the high mortality figures usually resulting from artificial feeding. With this purpose in view, we should carefully investigate different methods of feeding and adopt some more uniform plan for starting human beings in life; for diversity and not uniformity is now the rule. While inherited diseases contribute a certain proportion of the deaths which occur in infants, yet diversity of method in feeding is the most prolific source of disease in early infancy. The group of symptoms which for want of a better name is designated as difficult digestion occurs most frequently in the three periods in which the infant's digestion is likely to be tampered with,—namely, in the early weeks of life, when experiments are being made to determine what food will be best to start with; next, when, in addition to the irritation arising from the beginning of dentition, new articles of diet are added to the original food; and, thirdly, at the time of weaning, when there is often a sudden and entire change in the character of the food. The proper management of the first of these periods is of the greatest importance, because it is the time when the stomach is in its most active period of growth, and when the function of digestion is being established, and following the rule of functional establishment, is in a state of unstable equilibrium.

We should recognize the fact that the problem of substitute feeding is not a simple one. We cannot reiterate too often that the question which commonly is supposed to be a simple one, and the one which in the great majority of cases is alone considered,—namely, "Which food shall we give to the infant?"—is a misleading and insufficient one. The problem is a combination of factors of which the kind of food is only one, and I personally have long been convinced that the neglect to investigate thoroughly and carry out in detail the combination of these by no means insignificant general factors has had much to do with our failures in subsitute feeding in the past. It would seem, also, that the present is a most opportune time for raising a note of warning against allowing our enthusiasm over any one especial theory to warp our better judgment. There will surely be a reaction which will relegate to its proper place every theory built upon single factors of the problem before us, and which is actually doing harm by keeping in the background other theories which, each in its own sphere, as a significant part of a complete whole, may be of very great importance in the successful solution of the general problem. An error of oversight of one-eighth in a mathematical problem is not so great as one of one-fourth, but nevertheless the correcting of the greater error will not prevent an oversight of the smaller from completely destroying a correct result. Until lately it has been the quality of the food which has been monopolizing to too great a degree the attention of the medical profession. Then it was sterilization in feeding which became prominent. A German writer on substitute feeding has stated that the physiology and pathology of infantile digestion depend not on the chemical but on the biological character of the food. If we are not on our guard, this exaggeration of each single factor will prevail, and by its influence will blind us to much good work which in other directions has already been done, and which we cannot afford to ignore. Not that I would for a moment be understood to underrate the value of feeding an infant on a sterile food, for it has for years proved of very great benefit in my practice and that of others, but I predict that by just so much as we enhance the value of this one important part of the whole at the expense of the others, just so much farther shall we be from an intelligent comprehension of the whole subject. To feed an infant one month old with six ounces of acid cow's milk every four hours, no matter how thoroughly such a mixture has been sterilized, would be a radical offence against well-known anatomical and physiological laws. We should investigate and endeavor to copy, each in its turn, the various devices which nature makes use of, for we must admit that we are not in a position to improve on nature's method.

It is certainly wiser and more economical not to spare expense and trouble in arranging the infant's diet, for the period of active growth of an organ is the time when its function is readily weakened, and when once weakened, the digestive function is a prolific source of annoyance and expense in childhood and adolescence. Cheap foods and cheap methods of feeding, unless they are the best that can be procured, should not be tolerated in the early feeding of infants. We often, however, see a food recommended for a young infant because it is cheap and easily prepared, in spite of the fact that its well-known lack of nutritive ingredients should stamp it as unfit for use.

In discussing the treatment of disease we advocate what is best, without reference to what it costs, and then, in the special case where expense is an element which has to be taken into consideration, we endeavor to adapt our treatment to these considerations, and approach as nearly as possible to our first standard. In like manner I believe that we are doing wrong to the public if we allow ourselves to be handicapped in so difficult a question as infant feeding by the cry of expense. Infant feeding is an expense which is vital to the welfare of the human race, and we can, without being accused of extravagance, safely relegate to the province of the manufacturers of patent foods the recommending to the public of foods which if judged by the amount that is offered in bulk are cheap, but which when judged by their nutritive properties are extremely expensive.

Our scientific knowledge and clinical investigations have not yet enabled us to follow nature exactly, and we therefore have not yet obtained an ideal method of substitute feeding. We must, nevertheless, go as far as the present state of our knowledge will allow, thus gaining a little ground every year; and we must be especially careful not to be led astray by the fictitiously brilliant results which are reported from time to time in favor of certain foods. Instances are continually occurring where one food will fail and another, when substituted for it, will succeed, and yet these successes are merely temporary, and the disturbances of nutrition and mortality resulting from the use of various infant foods always remains far above those which occur from the use of human breast-milk.

SOURCE OF FOOD.—Having decided to substitute some food in place of woman's milk for the infant, we must decide from what source the elements of this food shall come. The food which approaches most nearly in every respect the product of the human mamma is that produced by the mammæ of other animals. The reason for this is that the food which all mammals provide for their offspring is an animal one, and consists of the same elements, although the mammary product of different animals varies in the percentage of these elements.

Assuming, then, that average human breast-milk is the safest standard for us to copy, we are impressed with the fact that although a vegetable diet would often seem far the easiest method of procuring nourishment for young infants, yet nature has persisted in providing an animal one. We should therefore be very careful not to introduce into our substitute diet a vegetable element, which, as judged by our standard, must be a foreign element. Milk is the food which our reason tells us should be given to the young infant, and a milk which will approach as nearly as possible to the average human milk.

The milk of various animals has from time to time been recommended as the best substitute for human milk, the recommendation being based on their analyses approaching more or less nearly the composition of human milk. The milk, however, of all animals has to be modified to correspond to human milk; and when we begin to modify, it is as easy to change the percentages of the different constituents to a great degree as to a small. The fact that the milk of any particular animal approaches in its analysis nearly to that of the human breast is not of much significance, other considerations being far more important; and it is most important of all that we should use one which can be obtained easily by the people at large. This at once settles the question that it is the milk of the cow to which we must turn our attention, even though cow's milk may differ in its composition from human milk to a greater degree than does the milk of the ass or the mare, whose milk approaches, so far as is shown by analyses, most nearly of that of all animals to human milk. If, however, the ass and the mare should be employed for dairy purposes to the same extent that the cow has been, there is every reason to suppose that their milk might change in its composition and their comparatively undeveloped mammary glands increase in size, just as has been the case with the cow, an animal which for thousands of years has been used for the production of milk, and which probably did not in the beginning give such an over-production of the mammary secretion as is the case now. It is, then, from the public demand, and by breeding, that cows have been made to produce so much more milk than is necessary for the support of their young. Not only quantitative but qualitative differences exist in animals according to the development of their mammary glands; and, as Martiny has shown in his collection of statistics on this subject, the condition which determines the quantity and the quality of the milk depends on the development of the organ which produces it.

A further exemplification that cow's milk is practically the universal source of the substitute food-supply for infants in most civilized communities is the fact that the various foods, patent or not, all depend for their basis on cow's milk, and that without this addition of milk they would show but an insignificant percentage of many of the most important ingredients of the food. Logically we should not speak of the various foods as such, but merely as adjuvants to cow's milk. If this is thoroughly understood, much misapprehension regarding the apparently successful results of innumerable foods will be done away with.

Another reason for using cow's milk in preference to all others is owing to the fact that the cow can be kept under more strict control than any other mammal.

THE COW.—Having chosen the cow for our primal milk-supply, we must next consider whether any special breed is better adapted than others for accomplishing our purpose. To do this we should first examine chemically and microscopically the elements of the milk of those breeds which can be employed best throughout the civilized world. It has been found that the finer breeds of cows from the Channel Islands are more liable, when transported from their home to countries where the climate is more severe, to contract diseases, such as tuberculosis, than are the animals represented by the Durham, Devon, Ayrshire, and Holstein breeds.

Among the breeds of cows which should be used for infant feeding at the farms connected with the laboratories are the following.

The **Durham, or Shorthorn,** represents the best type of cow for this purpose. She has great constitutional vigor, great capacity for food, a perfect digestion, a placid temperament, and yields a large quantity of rich milk, of which the analysis is as follows:

	Per cent.
Fat	4.04
Sugar	4.34
Proteids	4.17
Mineral matter	0.73
Total solids	13.28
Water	86.72
	100.00

Another breed, the **Devon,** has the same general characteristics as the Durham. The cows are gentle and vigorous, and give a moderate quantity of milk of medium quality, the analysis of which is as follows:

	Per cent.
Fat	4.09
Sugar	4.32
Proteids	4.04
Mineral matter	0.76
Total solids	13.21
Water	86.79
	100.00

Another breed is the **Ayrshire,** whose constitutional vigor is great, but whose temperament is nervous. The cows are not so hardy as the Durham, but are very free from disease. They yield a large supply of milk with the following analysis:

	Per cent.
Fat	3.89
Sugar	4.41
Proteids	4.01
Mineral matter	0.73
Total solids	13.04
Water	86.96
	100.00

Another breed which is of a thorough dairy type is called the **Holstein-Friesian**. This cow represents the most perfect milking animal known, having every characteristic of a cow suitable for laboratory purposes, but her milk is so light in its total solids that it is not so profitable as the other breeds. She yields a larger quantity of milk than any other known breed, although the analysis shows it to be poorer in quality. The following is the analysis:

	Per cent.
Fat	2.88
Sugar	4.33
Proteids	3.99
Mineral matter	0.74
Total solids	11.94
Water	88.06
	100.00

Another breed is called the **Brown Swiss** grade. The cows are very vigorous, healthy, and docile, and yield a fair supply of milk of about the richness of the Devon, the analysis of which is as follows:

	Per cent.
Fat	4.00
Sugar	4.30
Proteids	4.00
Mineral matter	0.76
Total solids	13.06
Water	86.94
	100.00

Finally, we can make use of the little **Bretonne** cow, known all over Europe as the "cow for the family." Cows of this breed have all the characteristics of the good domestic cow which have already been mentioned. They produce a medium amount of milk, large, however, in proportion to their size.

Some of the marks which distinguish the breeds of cows best adapted for infant feeding are: (*a*) constitutional vigor, (*b*) adaptability to acclimatization, (*c*) notable ability to raise their young, (*d*) freedom from intense inbreeding, (*e*) a distinctly emulsified fat in the milk, (*f*) a preponderance in the fats of the fixed glycerides over the volatile glycerides. The volatile glycerides do not exist in the mammae, but are formed in the milk soon after the milking. In some breeds, as in those of the Channel Islands, this change occurs more quickly than in others. Such breeds as the Jersey, Guernsey, and any others in which intense inbreeding has been carried on and in which acclimatization has not been perfected, should not be used for infants and young children. These breeds, of course, do not represent all of those available for substitute feeding, for we may mention many others equally good each in its country. For example, the Kerry of Ireland, the Red Polled of England, the Dutch Belted and the Flemish, also the Flamande and the Cotentine of France, the Norman breed of Normandy, besides the Brown Swiss just spoken of, and the Simmenthal, sometimes called Bernese, of Switzerland, also the Chianina of Italy, and the Allgauer of Germany. The native cow of this country, the "Red Cow," through many generations of neglect and exposure in winter, has undoubtedly acquired an impaired digestion and does not respond readily to appropriate changes of food.

CARE OF THE COW.—A cow whose milk is to be used for purposes of infant feeding should be properly housed and well cared for, as the domestic cow is an animal peculiarly sensitive to her surroundings, and her product is correspondingly liable to be thrown out of equilibrium. The milk product of a herd of healthy cows is much less liable to the variations so injurious to the infant's digestion than is the milk of any one cow. It is especially to be noticed how much easier it is by proper care to control exaggerated nervous influences upon the cow's product than upon the woman's. This at once suggests to us the question, Where and how shall cows be taken care of?

The cow is a sensitive animal, easily yielding to conditions good or bad in which she is placed. She is liable to contract diseases communicable to man, and especially to infants, and she is a ready vehicle for the transmission of obscure and often untraceable affections of a septic character. Her surroundings are such as to favor infection, and her attendants are often the means of conveying to the milk many of the transmissible diseases, such as scarlet fever, diphtheria, and tuberculosis.

The ordinary cow is allowed to range over wide pastures which are sometimes overflushed with herbage and sometimes parched by drought, and which nearly always contain noxious weeds, which she seems eagerly to seek. Again, she is forced to drink from stagnant pools and polluted streams, and at other times suffers for want of water for many hours together. She is also frequently exposed to storms. Cows cared for in this way are not those which provide the best milk for substitute feeding. These are the adverse conditions which surround the ordinary cow during the summer. In the winter she is crowded in the stifling atmosphere of a close barn with the manure of the whole winter kept underneath the floor on which she stands. Her head is usually confined in a narrow stall. The fodder intended for the winter's supply is kept above her head, and is continuously contaminated by the foul odors of the barn. She is turned out to the watering-trough at periodical intervals. Thus she cannot be said to be cared for in a manner conducive to the equable function of her mammary gland.

For cows to be used for the purpose of infant feeding a barn is needed where each cow shall have at least twelve hundred cubic feet of fresh air. The food should be kept where it cannot be contaminated. The manure should be as carefully removed from the barn as if it were a human dwelling. The cow should have freedom for her head and limbs in wide stalls. Large, dry, sunny exercise-yards should be provided for her. Her food should always be brought to her and selected with great care. Pure water should be provided, and suitable cups or troughs containing running water should be in her stall. The bedding should be fresh and free from mould or from any soil productive of bacterial growth. Methods should be used to get rid of all the usual foul odors and free ammonia so commonly produced in barns. Cows should be carefully guarded against fright, the worrying of dogs, and unusual excitements of all kinds, which cause serious disturbance of the lacteal functions of domesticated cows, in contradistinction to those of cows in a more natural condition, as, for instance, the cows in a semi-wild state on the plains of Montana, Texas, Australia, and the Pampas of South America. Excitement does not apparently injure the lactation of these cows, while it inevitably throws out of equilibrium the milk of the well-cared-for dairy cow. If the same care should be applied to regulating the woman's life as can be employed in the barn, we should encounter fewer difficulties in human breast-feeding. The feeding of the cows should have for its object the production of an even, nutritious, digestible milk and the careful avoidance of overstimulation of the lacteal secretion. The exact chemical analysis of any one ration used for feeding cows for our purpose must be carefully considered in accordance with the ratio of the digestible nutrients of the food, and this must of course be arranged practically from the recognized food tables. A great variety of food is necessary in feeding cows, but in the transition from green foods to dry, or the reverse, much care is needed to graduate the change, as disturbance in the equilibrium of the mammary gland is rapidly followed by injurious effects on the consumer. In past times, before I could rely as I do now on this carefully managed change of rations, the spring of the year with its flush pasturage and the fresh grass following the autumn rains were fruitful sources of infantile digestive disturbance in my nursery practice.

All these links in the chain which constitutes a successful substitute feeding are of great importance. The cows must be kept clean by grooming and the necessary washing, the precaution always being taken to rub the moistened parts dry. The milkers should be dressed in clean, freshly sterilized white suits and caps. Their hands and arms should be thoroughly scrubbed before milking. The hands in milking should be kept dry. The milking-stools should be made of metal so that they can be sterilized. The milk should be drawn with some force, simulating the action of the calf, and at each milking every drop of milk should be drawn out. The milk should be drawn into sterilized pails and carried immediately from the barn to the milk-house, which should be a sufficient distance from the barn to be free from odors. No means yet known to science can prevent some few bacteria coming into the milk during the milking-time, though it is possible to reduce the number so greatly as to make the milk practically sterile for the purpose of infant feeding, particularly if the second half of the product of the udder alone is used and milked into sterile tubes. The first half probably contains many bacteria, which, entering from without, have reached the lower portion of the teat.

The major part of the bacteria present in the milk are such as cause the usual acid fermentation which we recognize in the common souring of milk, but there are many species of bacteria which ought to be prevented from gaining access to the milk, arising from mouldy hay, straw, or fodder, partially decayed roots, and the natural decay of the wood-work of the barn and adjoining buildings. These latter varieties, which are found to be especially inimical to the preparation of substitute foods, cause in some cases the alkaline fermentation and other abnormal conditions of milk. Every barn apparently has its own set of bacteria, and the flora in America do not exactly resemble the analogous European species which have so often been described.

The bacteria which are found in cow's milk do not necessarily come from external sources, whether they be of the cow herself or of her surroundings, but may also come from some part of the milk tract between the udder and the end of the teat. These conclusions, it may be said, are made with reference to healthy cows.

Infectious mammitis, to some extent, seems clearly to be carried by the hands of the milkers from cow to cow. This also points to the fact that bacteria may find their way to the ducts through the teats.

TUBERCULIN TEST OF COWS.—It is very important that certain precautions should be taken to prevent the use of cows which are affected with tuberculosis. It is probable that three per cent. of the cows whose milk is used for food are tuberculous. Where tuberculosis is developed to such a degree in the cow as to be dangerous to the consumer of the milk, the disease can be usually detected by a skilful veterinarian by means of the physical examination which is employed in cows. But, as it is a disputed question at present as to when the milk of a tuberculous cow becomes affected, it is wiser to adopt all measures of precaution known to science. Of these measures the one which is most efficacious in detecting even the incipient stages of tuberculosis is the "tuberculin test." All the cows in use at the farms of the milk-laboratories are subjected to this test, and unless a negative reaction is obtained they are isolated from the rest of the herd.

CARE OF THE MILK.—After the cows are milked, the milk should be carried quickly from the cow to the milk-house, which should be at least a hundred yards from the barn and completely isolated from all other buildings. To prevent the milkers from going into the milk-room, the milk at the farms connected with the milk-laboratories is poured by means of a block-tin pipe through the wall of the milk-room into a large ice-lined block-tin tank, which is also the mixer for the milk of the entire herd. In the space of four minutes, by means of an ice-jacket, the milk is cooled from 33.88° C. (93° F.) to below 4.44° C. (40° F.). This is to rapidly remove the heat, which is conducive to bacterial growth. The milk passes through eight thicknesses of sterilized gauze on its way to the tank. The milk-room is practically clean from a bacteriological stand-point, for the walls and floor are kept wet with clean water, and all dust is excluded. The air which enters the milk-room is washed with sterilized water as it enters. Part of the milk is then passed through the separator, while part is drawn into sterile jars for transportation. No one is allowed to enter the milk-room except the man in charge, and he is carefully trained to be absolutely clean in person, taking special care of his hands, face, and hair. While in the milk-room he wears freshly sterilized white clothes. In the milk-room the jars are sealed, packed in ice, and in a few hours delivered at the laboratory.

After milk has been treated with these precautions, I have had repeated bacteriological examinations made on its arrival at the laboratory, with the uniform result that it has proved to be comparatively sterile, and at times it has contained either no colonies of bacteria or only one or two.

No antiseptic can, without danger to the infant, be used about the cow, while all the mechanical devices heretofore tried to take the place of manual milking have inevitably tended to impair the lacteal function of the udder.

CHEMISTRY OF COW'S MILK.—We have spoken of milk in general, and of certain points in connection with the chemistry of human milk in considering the subject of maternal feeding. In *indirect substitute feeding*, which involves a consideration of the principles governing the modification of cow's milk, it is of the first importance that the physician should acquaint himself with the chemical, physiological, and bacteriological characteristics of the food with which he is dealing. The failure to appreciate the importance of such a knowledge is responsible for much of the ill-founded criticisms of those who oppose the methods of percentage feeding, and the failure to obtain satisfactory results by those who have attempted to adopt percentage feeding without understanding its fundamental principles. It is, therefore, well worth our while to consider in more or less detail certain points bearing on the chemistry of cow's milk.

AVERAGE ANALYSIS.—The following averages are from analyses by such well-known chemists as König, Forster, and others:

Reaction	Slightly acid.
Specific gravity	1029–1033
Water	86–87 per cent.
Total solids	14–13 "

	Per cent.
Fat	4.00
Sugar	4.50
Proteids	4.00
Total mineral matter	0.70
Chlorine	13.45
Sulphur	0.41
Phosphoric acid	27.98
Iron oxide and alumina	0.44
Lime	23.17
Magnesia	2.63
Potassium	53.00
Sodium	4.49

Reaction.—Perfectly fresh cow's milk is generally amphoteric in reaction, but on exposure to air becomes more and more acid, owing to the formation of lactic acid by the action of certain micro-organisms upon the milk-sugar. The relative proportion of the acidity and alkalinity of the amphoteric reaction varies in different cows and at different times during the period of lactation, and is undoubtedly influenced by the character of the food on which the cow is fed. Experiments seem to show that the milk drawn from cows fed on the better grasses in a half-ripe condition is much more alkaline than when the cows are fed on dry fodder and grain, which increase the acidity of the milk.

These experiments are of great interest as showing that not only can the product of the cow, so far as its reaction is concerned, be made to correspond to that of human beings by means of perfectly natural feeding and under perfectly normal conditions, but that this alkaline modification can be produced to such a degree that one-third of the milk is sufficient to destroy by its alkalinity the acidity of the remaining two-thirds. It is doubtful, however, if it is desirable to attempt to modify the reaction of milk for infant use by any such method of feeding.

The importance of the subject lies in the well-recognized fact that the infant's digestive functions have been from time immemorial better adapted to the digestion of an alkaline or a neutral fluid than of an acid one. Whether the moderately alkaline reaction of human milk is an important factor in the problem of infant feeding is a question which future investigation alone can completely prove, but with our present knowledge we are not prepared to dispense with even the least important of the many factors which make up this problem. At any rate, we should be very suspicious of a breast-milk which shows an acid reaction. In the preparation of an infant's food from cow's milk, according to the latest experiments by means of modification, the best results have been obtained by making the reaction of this food correspond to that of normal human milk. This, up to the present time, has been done best by the addition of an alkali, which is the only foreign element that it has been found necessary to employ.

As it is wise in preparing a mixture for substitute feeding to make such a mixture approach as closely as possible in both taste and reaction to woman's milk, Harrington's experiments made at my request (Table 39) with lime-water and ordinary cow's milk twenty-four hours old are important. Lime-water was the alkali used in these experiments because it is the most simple adjuvant which we can use for making cow's milk alkaline, the amount of lime contained in it being so small that its addition in even considerable quantity does not materially alter the amount of the total mineral matter. As small an amount as one-sixteenth part, when added to ordinary milk, will render it alkaline, so that for making an acid milk correspond in its reaction to woman's milk, lime-water is of great value, as it apparently does not produce any other changes in the milk. In addition to this, the taste of a mixture which is made from ordinary cow's milk, so as to correspond to the composition of woman's milk, is strikingly like that of woman's milk if it contain one-sixteenth part of lime-water.

Harrington has made an estimate by actual experiment of the amount of lime-water which is needed to produce an alkalinity in a mixture such as has been just mentioned which would correspond to the alkalinity of human milk. This table (Table 39) shows the results of his experiments.

TABLE 39.

Amount of Lime-Water in Mixture.	Reaction.
25 per cent	Strongly alkaline.
12.5 per cent	Still strongly alkaline.
6.25 per cent	Slightly but distinctly alkaline, and corresponding to woman's milk.

It must be remembered that these proportions of lime-water are those required for ordinary milk twenty-four hours old, a much smaller proportion being needed to produce the same results when the milk is collected and handled with the precautions insisted upon at the farm connected with the milk-laboratories.

Specific Gravity.—The specific gravity of cow's milk varies from 1.028 to 1.034, and does not differ materially from that of human milk.

Milk-Fat.—The fat of milk occurs as minutely divided globules, held in suspension in the milk-plasma, forming a fine emulsion. It is definitely proved that all of the fat occurring in the milk is contained within these globules, but whether the globules are purely fat is a disputed point. According to Storch, the globules are surrounded by a slimy substance called "stroma substance," which, by his analyses, is shown to be neither casein nor lactalbumin, but a nitrogenous material containing fourteen per cent. of nitrogen and a reducing substance on boiling with mineral acids. The fat is composed for the most part of the neutral palmitin, olein, and stearin, and the triglycerides, myristic, butyric, and caproic acids, with traces of other unimportant fatty acids and extractives. The percentage of *fat* in the average cow's milk and in the average human milk is practically the same, *i.e.*, about four per cent. The glycerides of the fatty acids composing the fat in both cow's milk and human milk have been determined, yet our chemical and clinical knowledge of the nutritive value and digestibility of these, separately or collectively, has not arrived at a point where we can practically make use of this knowledge, and we therefore direct our attention to regulating in a milk modification the percentage of the fat as a whole.

Fig. 51, page 204, a photomicrograph of a thin layer of milk, shows the minute globules of fat permeating the transparent medium. This fat is simply held in suspension, which enables us to separate it easily by mechanical means. It is, in fact, in the condition which marks the milk as an emulsion.

Milk-Plasma.—The milk-plasma is the fluid in which the fat globules are suspended. It contains the caseinogen, lactalbumin, lactoglobulin, milk-sugar, and various extractives, such as urea, creatin, creatinin, hypoxanthin, lecithin, cholesterin, citric acid, and certain mineral bodies and gases. Clinically, we cannot, in our present state of knowledge, state the significance of the presence of these bodies, though they undoubtedly are factors in the general metabolism of the body. The milk-sugar, caseinogen, and lactalbumin are, on the contrary, of the first importance, and their nature should be clearly understood.

Milk-Sugar or Lactose.—The sugar of milk in all mammals is of the variety called *milk-sugar* or *lactose*. It is a simple and uniform element to deal with. Its percentage in cow's milk is about 4.5, and in woman's milk about 7.

Regarding the kind of sugar which should be used in making up a substitute food, we have certain questions to consider which would seem to be important. Cane-sugar has been, and still is, a favorite form with which to regulate this part of the solid constituents of the food. The reasons given for using it have been its preservative qualities, as seen in the manufacture of condensed milk, and the theory that it is not liable to set up excessive so-called lactic acid fermentation, with its consequent disturbance of digestion, as has been supposed to be the case with milk-sugar. Cane-sugar in a concentrated form, as it is found in condensed milk, seems to act as a preservative, but when it is diluted, as in its administration to the infant, cane-sugar ferments very readily, and in this respect has no advantage over milk-sugar. Reasoning from analogy, we should say that as milk-sugar is the only form of sugar found in the milk of mammals, it is there for some good purpose, and that it is needed for the accomplishment of some process which takes place after the food has been swallowed. Both cane-sugar and milk-sugar are converted into glucose either in the intestine or in the process of absorption from the intestine. There seems, however, to be some difference in the degree to which they can be used for purposes of nutrition before they are converted into glucose. So far as is known, whether in plants or in animals, cane-sugar is merely a reserve, and cannot be used directly for nutrition. Milk-sugar, on the other hand, is probably not merely a reserve, but may possibly be utilized in the economy also for nutrition. Thus, Bernard has shown that seven grains of milk-sugar dissolved in an ounce of water could be injected under the skin of a rabbit without the subsequent appearance of sugar in the urine, while under the same conditions and in the same amount cane-sugar was found to be eliminated as foreign matter by the kidneys.

Milk-sugar undergoes no direct alcoholic fermentation except when exposed to certain unusual ferments, but it changes readily to lactic acid in the presence of nitrogenous ferments. The lactic acid fermentation may be checked by heating the milk. Cane-sugar, on the other hand, easily undergoes alcoholic fermentation, but changes to lactic acid less readily than milk-sugar. Cane-sugar, moreover, takes on the butyric acid fermentation more readily than does milk-sugar. The *bacillus lactis aërogenes* (Escherich) is present in normal digestion, and acts on the milk-sugar to produce an organic acid which drives out the more noxious forms of bacteria, which by their presence would interfere with normal digestion. When milk-sugar is converted into glucose and galactose, we physiologically have a gradual conversion into lactic acid, which may aid in the digestion of the proteids, thus giving us a very valuable addition to the means at our command for rendering modified cow's milk digestible.

Jeffries says, in reference to the different actions of the various kinds of sugar in the digestive tract, that it is important to note that starch, dextrin, inulin, cane-sugar, and dextrose afford material for the butyric acid fermentation, while milk-sugar does this only after completed hydration.

Escherich, in speaking of Brieger's bacillus, says, "Milk is coagulated with sour reaction first after several days (eight to ten) at the body temperature. With exclusion of air this bacillus cannot grow either in milk or milk-sugar solution, but will in grape-sugar.

We thus see that the milk-sugar offers less danger of the butyric acid ferment, which we know makes much trouble at times in the body, and that under certain conditions of the intestine it should be exempt from the assaults of Brieger's bacillus.

When we consider that by means of heat we can practically put an end to the lactic acid fermentation, which may have begun to act upon the milk before it enters the stomach, it would seem that we are justified, on both physiological and bacteriological grounds, in using the same animal sugar in substitute feeding that is found in the infant's natural food, instead of introducing a vegetable sugar, which in milk is a foreign element.

The dangers from lactic acid are, at any rate, much exaggerated by writers on this subject.

Proteids.—The proteids of normal human milk belong to the nucleoalbumins, and have quite a wide range in their variation; still, it is now well recognized that their average normal total percentage is very much below that of cow's milk. Assuming that the percentage of proteids in human milk is 1.5, or between 1 and 2, it can be stated that the relation of the percentage of the proteids in cow's milk and in human milk is as 4 to 1.5.

The proteids represent the nitrogenous elements of milk They consist of two distinct elements, *caseinogen* and *lactalbumin* (for clinical purposes it is not necessary to consider the lactoglobulin as separate from the lactalbumin), which together make up the total percentage of proteids in both woman's and cow's milk. These two elements differ greatly, however, in their relative proportions to each other in the two milks. This difference is shown in the following figures taken from König:

	Woman's Milk. Per Cent.	Cow's Milk. Per Cent.
Caseinogen	0.59	2.88
Lactalbumin	1.23	0.53
Total proteids	1.82	3.41

According to the observations of other investigators the proportion of lactalbumin in whey is one per cent. (Bulletin 28, United States Department of Agriculture), which gives a higher percentage than in the figures quoted from König. Another analysis of whey by Hammarsten gives the percentage of lactalbumin as 0.86.

Thus it is seen that of the total nitrogenous constituents of milk which are classed under the general term proteids, and of which the caseinogen and lactalbumin are parts, the coagulable proteids or caseinogen in cow's milk are proportionately larger in amount than in human milk, so that under the same conditions a larger curd will be formed with the former than with the latter.

The question whether the casein in all milk is the same, or whether there are several different caseins according to the difference in the species of mammals, has not been determined.

The casein is precipitated from its solutions by acids, but the precipitation is retarded by the presence of neutral salts. It is soluble in small excess of hydrochloric acid, but is again precipitated by a large excess.

This acid coagulation of milk is to be clearly distinguished from the coagulation which takes place as a result of the action of the rennet ferment, by which the milk, when fresh, coagulates into casein and "sweet whey" without any change in its reaction.

Mineral Matter.—The mineral matter of cow's milk has been analyzed with comparative care and success. The total salts obtained by the analyses of König was 7.1 parts in 1000 parts, or 0.71 per cent. According to Söldner, the potassium, sodium, and chlorine are found in the same quantities in whole milk as in milk-serum. Of the total phosphoric acid, 36 to 56 per cent., and of the lime, 53 to 72 per cent., is not in solution. A part of the lime is combined with the caseinogen; the remainder is found united with the phosphoric acid as a mixture of dicalcium and tricalcium phosphate, which is kept dissolved or suspended by the caseinogen. The bases are in excess of the mineral acids in the milk-serum, and the excess of the former is combined with organic acids. At present the percentage of the mineral matter in cow's milk does not enter into the clinical modification of milk, but a more extended knowledge of the subject may in the future be found to be of importance.

The differences between the constituents of the mineral matter of human milk and of that of cow's milk are as follows: in cow's milk there are more lime, magnesia, potassium, much more phosphoric acid, and less chlorine and sulphur.

Water.—There is about one per cent. less of water in cow's milk than in human milk. Chemical analyses invariably show so large an amount of water in human milk that it is evident that the infant is intended to take, and can best assimilate, a very dilute food. We must bear this fact in mind in preparing a substitute food, as the precaution of supplying a thoroughly diluted food is of extreme importance in managing the infant's feeding both in health and in disease.

Total Solids.—There is about one per cent. more of total solids in cow's milk than in human milk. These solids in the milk are held partly in solution, partly in semi-solution, and partly in suspension.

ATTENUANTS.—So much has been said about, and so many physicians are in favor of, diluting cow's milk with such attenuants as barley-water and other cereal waters, that a clear understanding should be had as to the true position which such attenuation should hold in infant feeding.

One of the objects which physicians expect to attain with cereal attenuants is that in some mechanical way the attenuant breaks up the coagulated proteids into finer particles than when the milk is simply diluted with water or lime-water or solutions of sugar. Barley-water seems to be the best attenuant for this purpose, and in comparing the coagulum obtained by the dilution made with this cereal with that made with water, lime-water, or sugar-water, it is found to be somewhat finer in the former than in the latter. The barley-water should be made as directed on page 239. A decoction of barley-water made in this way contains rather less than one per cent. of starch. This proportion of starch does not add materially to the nutritive value of the mixture, and must merely be looked upon as a foreign element which is never found in human milk. If, as has also been recommended by certain physicians, this starch in the cereal attenuant is dextrinized, the attenuant becomes a solution of sugar, and has no more mechanical effect on the coagulum than sugar in water.

The use of cereal attenuants, excepting when they have a higher starch percentage for purposes of nutrition, is chiefly confined to the practice of those physicians who have not made an extended study of the finer possibilities comprehended in laboratory modification, and have failed to appreciate that the older methods of treating the coagulum of cow's milk are not needed in the newer. Human milk never contains starch; the amylolytic function of the infant is not fully developed in the early months of life, and should not be taxed in the process of its development.

The small additional mechanical effect of cereals on the coagulum is insignificant in comparison with the more rational methods of treating the coagulum ; these are truths which the medical profession will acknowledge when the laboratory system is better understood and laboratory methods still further perfected.

The newer and more rational methods just referred to of dealing with the coagulum are not only to dilute sufficiently with water, but so to treat the total proteids in the mixture that the relative proportion of lactalbumin and caseinogen shall approach as nearly as possible to that which is supposed to exist in the proteids of human milk. In this way the proportion of caseinogen being very much lessened, the resulting coagulum of casein will be much smaller, and practically so fine that any mechanical attenuant becomes useless.

If cow's milk is diluted with simple water 1 part to 3, the resulting coagulum is a little larger than the coagulum of human milk. If, however, the dilution is 1 to 4, the resulting coagulum is finer than that of human milk. In either case, however, the size of the coagulum must differ still further if the high percentage of caseinogen in the cow's milk proteid is reduced to the relative caseinogen percentage of human milk proteid, according to the total proteid of the mixture and the lactalbumin correspondingly increased.

Cow's Milk as compared with Woman's Milk.—We may in concluding the subject of the chemistry of cow's milk summarize the principal points of difference between cow's milk and human milk in the following table:

TABLE 40.

	Woman's milk directly from the breast.	Cow's milk as ordinarily received, about twenty-four hours old.
Reaction	Amphoteric. (More alkaline than acid.)	Slightly acid.
Water	87 to 88 per cent.	86 to 87 per cent.
Mineral matter	0.20 per cent.	0.70 per cent.
Total solids	13 to 12 per cent.	14 to 13 per cent.
Fats	4.00 per cent. (relatively poor in fatty acid.)	4.00 per cent.
Milk-sugar	7.00 per cent.	4.50 per cent.
Proteids	1.50 per cent.	4.00 per cent.
Caseinogen (König)	0.59 per cent.	2.88 per cent.
Lactalbumin (König)	1.23 per cent.	0.53 per cent.
Coagulable proteids	Small proportionally.	Large proportionally.
Coagulation of proteids by acids and salts	With greater difficulty. Curds small and flocculent.	With less difficulty. Curds large and tenacious.
Coagulation of proteids by rennet	Does not coagulate regularly.	Coagulates readily.
Action of gastric juice	Proteids precipitated but easily dissolved in excess of the gastric juice.	Proteids precipitated but dissolved less readily.

BACTERIOLOGY OF COW'S MILK.—A few matters concerning the bacteriology of cow's milk can best be considered in connection with the subject of substitute feeding. Respecting this question Dr. J. A. Jeffries very aptly remarks that "it is a curious fact that, while older people are chiefly fed on sterilized food,—that is, cooked food,—infants are fed on food peculiarly adapted by its composition and fluid state to offer a home for bacteria."

It is manifestly very important to use a milk for modification which is as free as possible from bacteria. In the milk commonly used in cities the number of bacteria to the cubic centimetre amounts to a million or more. Ten thousand bacteria per cubic centimetre is considered the maximum for good milk. In the year 1899 a daily examination of the milk produced by one of the farms connected with the milk laboratory in Philadelphia was made at the Pepper Laboratory. The average for 334 days was 1530 bacteria per cubic centimetre; and this number fell in the autumn and winter to 1150 to 1195 per cubic centimetre. These figures show how, with especial care at the farms, milk can be radically changed in regard to bacteriology.

In some experiments made by Jeffries agar-agar cultures were made before and after the different fluids were sterilized, and the colonies of bacteria were counted. His results coincide with those of previous experimenters,—namely, that steaming for fifteen minutes is sufficient to kill the developed bacteria, while a second steaming is necessary for complete sterilization. Out of one hundred and twenty lots of milk steamed but once, all but four or five showed distinct signs of change within a month, while the majority of those steamed twice did not change at all.

Jeffries's experiments also show that spores develop slowly, and, indeed, rarely form in milk, which, as he says, is an excellent medium for growth, while spore-formation among bacteria, like seeding among higher plants, is a phenomenon of impaired growth. He also explains the preservation of some of the milk steamed but once by the absence of any enduring spores from the start. In an article of very great interest and value to the practising physician, "On the Bacteria of the Alimentary Canal," Jeffries has reviewed, at my request, the work done by the various dacteriologists:

"Miller, De Barry, and Escherich have shown that living bacteria are to be found in the stomachs of men and animals, and the former author has also clearly proved that bacteria can pass through the stomach into the intestines and live for a considerable time. . . . Of the morphology and biology of the forms found in the stomach little is known. The field is a new one, and the species have not been sufficiently described to enable others to recognize them with certainty. Miller has found five kinds which give off carbonic dioxide and hydrogen gas, lactic, acetic, and butyric acids being formed. . . . Of the flora of the intestines much more is known than of that of the stomach. The researches of Brieger, Vignal, Stahl, and Escherich have now proved that a large number of species may occur in the fæces. Brieger isolated two new kinds: one a micrococcus, which turns grape- or cane-sugar into ethyl alcohol, with a trace of acetic acid; the other the well-known Brieger's bacillus. This species occurs in the fæces in vast numbers, ferments sugar, and decomposes albumins. Vignal isolated ten species from the fæces, six of these also being found in the mouth. Of these some produced acid fermentations and gas, but unfortunately they were not sufficiently studied to show their effects on digestion. . . . Escherich studied especially the fæces of infants, and found a large number of kinds of bacilli, among them a small bacillus capable of converting milk-sugar into lactic acid, carbonic dioxide and hydrogen gas being evolved, either in the presence or absence of air, a facultative anaërobic species, his *bacillus lactis aërogenes.* Escherich established, by the examination of a large series of cases, the fact that the kinds occurring in the fæces vary with the food,—that is, the intestinal contents. . . . Starting at birth with the sterile meconium, consisting of mucus, epithelium, and the like, infection by the mouth and rectum quickly occurs, and in a short time almost any form may be found, but chiefly such putrefying forms as *proteus vulgaris.*

"With the suckling of the infant and the substitution of the refuse of the milk and secretion of the digestive tract for the meconium, a sharp transition occurs. Instead of the generally distributed forms causing decomposition, only two kinds are regularly found, *bacillus lactis aërogenes* and Brieger's bacillus; the first chiefly in the upper parts of the intestine, the second in the lower parts. Passing on to the period of mixed diet, quite a number of forms appear, among them the *streptococcus coli gracilis*, the *putrefying green fluorescing*, a *tetrad coccus*, and *several kinds of yeast.* This brings us to the pith of the subject: Why are the flora so limited in the milk-eating infants and so diverse in others? What drives the forms found in the meconium out? That they can live there is clear, as shown by their presence the day before. Again, what prevents forms so common with meat diet from gaining a footing? It is not the milk alone, for milk is an almost universal food for bacteria, and all the kinds found in the intestines thrive in it.

"According to Escherich, the *bacillus lactis aërogenes* and the milk diet keep out the other forms.

"Formerly," continues Jeffries, "even before the action of ferments and putrefactive processes were clearly understood, the significance of this question was seen. The chyme is a mass admirably adapted for putrefaction or fermentation, yet ordinarily but little of either occurs. It is an alkaline or, as in the milk-fed, acid mixture, rich in albumins, fats, and the starch group, amply provided with water and warmth. Such a mixture outside the body at an equal temperature would quickly decompose. It was generally held that some preservative action was exerted by the digestive juices; Bidder's and Schmidt's dogs with biliary fistulæ were supposed to explain the whole. These dogs, deprived of their bile, became emaciated, and suffered from diarrhœa and decomposition of the intestinal contents. Thus it seemed clear that in the absence of the bile decomposition occurred,—that is, that the bile was a powerful germicide or germ-inhibitor. During the last few years, however, different results have been obtained in cases of biliary fistula. Rohmann's dogs did not suffer from diarrhœa or putrefaction in the intestines, hence it is clear that the bile is not the cause of prevention. The diarrhœa, if present, is due to the large amount of fat passed on to the lower intestines.

"Maly and Emich ascribed value to the bile acids, especially the taurocholic, basing their results on crude methods; and Lindenberger, really leaving the subject, attributed the action to the organic acids in combination with the bile.

"All this argument and belief in the decided germicidal action of the bile occurred in the face of the well-known fact that bile itself will decompose.

"From a bacteriological stand-point, Miller has shown that a ten per cent. solution of bile, if anything, favors growth. Macfadyen has studied bile, bile salts, and bile acids in varying strengths. The only positive results were got with the acids; these arrested the development of bacteria if sufficiently strong, especially taurocholic acid. Neither acid had much effect, and least of all on the forms causing putrefaction. *Proteus vulgaris* was only arrested by a strength of from one to two per cent. The pathogenic forms were arrested by a much smaller quantity, from one to one-half per mille.

"It is thus clear that other causes must be sought for. One of these is to be found in the lack of oxygen in the intestines, as pointed out by Escherich and strangely forgotten by others. There is certainly very little free oxygen in the chyme, if any; not only is it scarce in the food at the start, but is taken up by the chemical changes during digestion, and also by the intestines. This clearly must be a potent factor, for the majority of bacteria require a fair supply. Accordingly, many bacteria are found in the fæces which will grow in the air, as shortly stated by Macfadyen, and the mass of those isolated in the air are able to grow without it.

"This apparent contradiction, the absence of oxygen in the intestines, and the presence of both aërobic and anaërobic bacteria, is probably explained by the ability of the aërobic kinds to draw oxygen from oxyhæmoglobin. They thus breathe through the intestines, as it were, when in close contact with the walls, while the anaërobic kinds live in the mass of the chyme, and do not, so far as we know, reduce oxyhæmoglobin.

"Escherich, though he points out the absence of oxygen, does not seem to give it full value, or rather forgets the subject in treating of the action of his *lactic acid bacillus*. As before stated, this form is regularly found in great numbers in the upper part of the intestines of milk-fed children. Here it converts a considerable part of the milk-sugar into lactic acid, and thus prevents the other forms from growing,—most forms being susceptible to an acid reaction, and especially to the organic acids. The action of salicylic acid is known to all, and recent experiments, of which Macfadyen's (the last) are the best, show acetic, butyric, and lactic acids to be efficient germ-inhibitors in strengths of from one to one-half mille according to the species.

"In milk-fed infants another point is the comparative inability of bacteria to attack casein, so that the bacteria are literally starved.

"We may therefore conclude that the bile acids, lack of oxygen, lack of suitable albumins, and the presence of organic acids are the causes of immunity from the putrefying and fermenting kinds of bacteria to which we are exposed. Certain forms are probably limited by the lack of water,—that is, of a fluid state,—doing poorly if unable to swim freely about. It must not, however, be supposed that bacteria are scarce in the intestines; on the contrary, they form a large part of the dry substance of the fæces.

"The ferments act by the production of various acids, chiefly derived from the milk-sugar. In small amounts, as in the case of the *bacillus lactis aërogenes*, the acid seems to be of benefit, and certainly does no harm, as it regularly occurs in healthy breast-fed infants. In large amounts, however, it must tend to over-acidify the contents of the intestines and interfere with the action of the digestive fluids."

MILK-LABORATORIES AND PERCENTAGE FEEDING.—General Considerations.—When human milk that is suited to the individual infant cannot be obtained, or if obtained cannot be regulated by modification, it is desirable to substitute for it the combination of elements which such a human milk represents. To accomplish this we must have materials which, while closely resembling the elements of normal human milk, are easily obtained.

Physiological experiments on the mammary gland show that the albumin of the milk is not directly an exudation from the lymph-vessels supplying the mammary gland, but that it is actually modified in the gland itself. We thus see that the mammary gland, besides being an elaborator for infant nutrition, is also a modifier. This suggests to us that the modification of milk is not contrary to nature's method of preparing food for infants. Following, therefore, nature closely, we have learned that the proper modification of absolutely pure and fresh milk is the vital principle which should underlie our efforts to perfect a substitute food.

The infant at the breast receives for its nutriment a fluid which is fresh, sterile, amphoteric, or faintly alkaline, which has a temperature of 36.7°–37.8° C. (98°–100° F.), furnished in an amount proportionate to the age and size of the consumer. It is this fluid which we have to copy in every possible detail when we undertake to prepare a substitute food. We should also consider as foreign matter, to be carefully avoided, any element which we know is not to be found in the milk we are copying.

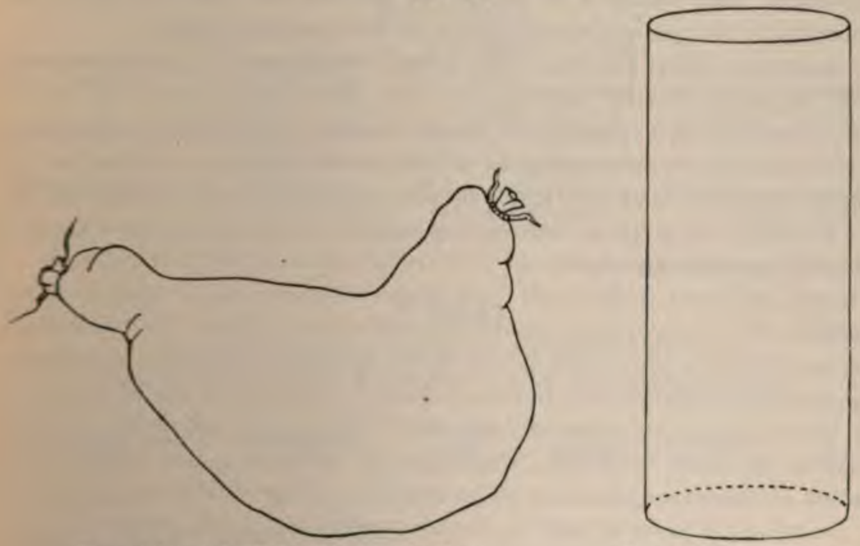
The analyses of human milk teach us that there is a great capacity in different infants to assimilate a variety of proportions of the same nutritive elements. In all probability the infant needs a variety in its food somewhat to the same extent as does the adult. In order, therefore, to copy nature closely, we must have some means of preparing a food not only for the many but for the individual, and when introducing new methods for preparing a substitute food we must recognize the necessity of providing for many prescription possibilities. In this busy age of scientific rational medicine physicians all over the world demand, first, means of saving time, and second, exact methods of work, which in themselves soon become time-savers. In every branch of our art the tendency is growing year by year to systematize the detailed and laborious work of the individual for the common practical use of the profession at large. The subject of substitute feeding should be reduced to a more exact system, and an effort should be made to rescue this important branch of pediatrics from the pretensions of the owners of proprietary foods and the hands of ignorant nurses. With this end in view, I have given my professional assistance to the establishment of a system of *milk-laboratories* where the materials used shall be clean, sterile, and exact in their percentages. These laboratories have been placed under the control of educated, intelligent men in whom we have the same confidence that we have conceded to the pharmacist, and we can write directions for infants' foods and send them to these laboratories just as, in the treatment of disease, we write our prescriptions for the division of one drug or the combination of several. As the pharmacist has nothing to do with the various methods of treating disease, so the milk-modifier is simply required to carry out the directions and ideas of the physician. No special school of medicine need be represented. No special method of feeding need be undertaken. An opportunity has, however, for the first time in the history of medicine, been presented for the physician to carry out his own methods, and these methods for the first time to be judged on a fair basis. In this way only can each clinical observer, when lacking in success, be sure that it is the fault of the food he is giving, and not because the food has varied from what he supposed he had ordered.

In quite a number of cases rather gross changes in the percentages of the different elements of the milk may be sufficient for the range of the individual digestion and for the nutrition of the especial case, but it has been my experience with a very large number of infants whose vitality was low and whose malnutrition was excessive, to find that the lives of these infants could only be preserved by gradual and minute changes in the percentages and combinations of the milk-elements. We therefore cannot be too particular in assuring ourselves that we are using a milk-modification which is as precise as any knowledge up to the present time has made it possible to be. Even slight changes, therefore, in the percentages of the three important elements of milk of which we have most accurate knowledge—namely, the fat, the sugar, and the proteids—are of real value in the management of the digestion and nutrition of the infant, and these changes are often necessary day by day as well as month by month. With this fact impressed upon us, we can well see that no one mixture will in all cases prove successful, but that a great variety in the percentages of the different elements of the milk will be needed in substitute feeding just as they already exist in maternal feeding. This explains the diversity of results obtained in the past with the same food by different practitioners. Regarding the subject from this point of view, it is evident that there is no especial combination of percentages for the especial age of the infant, for the proper nutritive value of the food given to an individual infant must be adapted to that infant's special stage of development and to its special powers of digesting certain percentages and combinations of percentages. It has been frequently observed that an improper modification for the individual may do much harm and may lead to something more than indigestion,—namely, to such diseases of nutrition as infantile atrophy, scorbutus, and rhachitis,—so that it is exceedingly important that the physician should understand the exact modification called for in each case as thoroughly as he would the treatment of any disease in the individual.

The means for prescribing a diversity in the elements of milk, according to the idiosyncrasy of the digestion we are dealing with, is supplied by a milk-laboratory equipped with special machinery and controlled by educated milk-modifiers. Purity of the original material is the first object to be attained. This material, milk, should be obtained from cows bred, fed, and cared for in the manner just described, and, in order to insure absolute uniformity in the methods, untiring vigilance must be used in the supervision of the farm, cows, and milk-house, and in the transportation of the milk from the farm to the laboratory. It is also necessary that the cows should be under the medical supervision of a skilled veterinary surgeon. These are all questions which to my mind have been definitely decided, but which now need time and attention devoted to them to insure their being systematically carried out. As in all other advances which are made in practical medicine, so also in this one it is well to adopt at once a high standard of work and to demand everything that can in any way tend to perfection. We may not always be successful in carrying out all the details, but, until we are, perfection will not be reached. It must, then, be borne in mind and understood that each link of that chain is of vital importance, because, if broken, the value of the whole chain may be lost. One end of this chain is at the milk-farm. Starting in the stall of the cow it passes on to the milk-house, from the milk-house to the milk-laboratory, and from the laboratory it should be carried unbroken and intact to the infant consumer.

APPARATUS FOR FEEDING.—Human ingenuity has not yet been able to devise anything which approaches the perfection of nature's apparatus for feeding, and the best that we can do to offset this complex mechanism is to adopt that which is exactly the reverse,—namely, an apparatus of absolute simplicity,—and thus combat the tendency to fermentation by preventing, through perfect cleanliness, the apparatus from becoming a source of fermentation. To accomplish this object the receptacle from which the infant is to be fed should be made of glass, in the form which will enable it to be most easily cleansed, and, as in the future the question of transportation will undoubtedly be a grave one, the receptacle should be such that it can be adapted to transit and not easily broken. For this purpose what are practically test-tubes fulfil these indications best. These tubes have open mouths larger than those usually provided in the ordinary nursing-bottle, and, having no angles, are readily cleansed. The artificial receptacle is not self-regulating, and hence we must determine the amount of food in bulk which nature provides for the average infant at different ages, and from these average figures deduce the proper amount for the especial infant. The feeding-tubes are graduated for the more important periods of growth, for the purpose of continually impressing upon the mother and nurse what the physician often has the opportunity of telling them only at the beginning of the nursing period,—namely, that the error is in giving too much food rather than too little, an error, also, which naturally results when, as is commonly the case, the usual eight-ounce nursing-bottle is provided as the receptacle at the very beginning of infantile life.

Fig 46



Stomach from infant 5 days old; capacity 25 c.c.
(Natural size.)

Glass cylinder, capacity 25 c.c.
(Natural size.)

I have found that I can easily convince most mothers of the mistaken zeal of nurses who advocate giving the young infant large amounts of food, by showing them the size of the infant's stomach at birth and comparing this small tube which corresponds to the stomach's capacity with an ounce nursing-bottle.

Nipples.—A nipple made of fine soft rubber adapted to the es infant as to its size and the holes for the milk is substituted for the n nal nipple. These rubber nipples should be large enough to be t inside out and carefully cleansed after each feeding. They shoul boiled after being used, and kept in cold distilled water with a little in it. They should be renewed frequently, the oftener the better: erably a new one should replace the old one three times a week. be found that the rubber nipple has to be adapted to the taste of the cial infant, and that it often has to be changed as to its size, texture holes before the infant is satisfied with it and sucks satisfactorily fro

Intervals of Feeding.—The intervals between the feedings given should also be adopted in substitute feeding, but the amount of fo be given now becomes a prominent feature in the division of the amount of food which it is proper to give in the twenty-four h according to the age and development of the individual infant.

Amount at Each Feeding.—The infant's weight and its gastric ca quite frequently do not correspond. Yet there seems to be no that the weight is a condition to which marked consideration shou given when we are attempting to determine so difficult a question proper amount of food to be given at each meal in the early months The amount to be given at each feeding must be carefully regulated ac ing to the gastric capacity.

TABLE 41.

General Rules for Feeding during the First Year.

The day feedings are supposed to begin at 6 A.M. and to end at 10 P.M.

Age.	Intervals, hours.	Number of Feedings in 24 hours.	Number of Night Feedings.	Amount at each Feeding.		Total Amount hours.	
				Cubic Centimetres.	Ounces.	Cubic Centimetres.	C
1 week....	2	10	1	30	1	300	
2 weeks...	2	10	1	45	1½	450	
4 weeks...	2	9	1	75	2½	675	
6 weeks...	2½	8	1	90	3	720	
8 weeks...	2½	8	1	100	3¼	800	
3 months..	2½	7	0	120	4	840	
4 months..	2½	7	0	135	4½	945	
5 months..	3	6	0	165	5½	990	
6 months..	3	6	0	175	5¾	1050	
7 months..	3	6	0	190	6¼	1140	
8 months..	3	6	0	210	7	1260	
9 months..	3	6	0	210	7	1260	
10 months..	3	5	0	255	8½	1275	
11 months..	3	5	0	265	8¾	1325	
12 months..	3	5	0	270	9	1350	

The above table shows how the intervals of feeding and the an of food to be given should correspond to the gastric capacity at diff periods of the first year. It is so important to avoid stretching so easily distensible an organ as the stomach that it is wiser to give too little rather than too much food in the early days of life, and then gradually increase the amount if the infant cries from hunger.

The first month being the most critical period for the infant's nutrition, as it is the time when the equilibrium of its metabolism is being established and its chance for life is least, especial interest should be attached to the series of careful investigations made at the Children's Hospital in St. Petersburg by Ssnitkin to determine the amount of food which should be given in the first thirty days of life. As the result of these investigations he deduces the rule, "the greater the weight the greater the gastric capacity." Ssnitkin's general results show that *one-one-hundredth of the initial weight should be taken as the figure with which to begin the computation, and to this should be added one gramme for each day of life.*

Illustration of Ssnitkin's rule to aid in adjusting the food to especially difficult cases in the first thirty days.

	Amount at each Feeding.		
Initial Weight.	Early Days.	At 15 Days.	At 30 days.
3000 grammes......	30 grammes. (About 1 ounce.)	30 + 15 = 45 grammes. (About 1½ ounces.)	30 + 30 = 60 grammes. (About 2 ounces.)
4500 grammes......	45 grammes. (About 1½ ounces.)	45 + 15 = 60 grammes. (About 2 ounces.)	45 + 30 = 75 grammes. (About 2½ ounces.)
6000 grammes......	60 grammes. (About 2 ounces.)	60 + 15 = 75 grammes. (About 2½ ounces.)	60 + 30 = 90 grammes. (About 3 ounces.)

It is wiser always to accomplish first the proper digestion of the food, even if there is no gain in weight, and then, when once the infant is digesting well, to increase the amount of the percentages of the different elements. At times when the infant is digesting well, and even gaining, it will suddenly cry so hard and with such evident hunger, that an immediate increase in the amount of its food is not only indicated but demanded, no matter what its age or weight. In these cases the stomach has probably grown rapidly and out of its normal proportion to the age and size of the child, and a larger supply of food is what is needed.

THE MILK-LABORATORY.—As milk-laboratories are now so well known, and have for some years been established all over the United States and in Canada and London, they need not be described in great detail.

A milk-laboratory should be a place to which the milk should be delivered from such farms and under such precautions as have already been described; it should be in a central locality, accessible to as many people as possible, and used *exclusively* to prepare ,from the prescriptions of physicians alone, any modification of milk which may be called for.

As milk is one of the best means for the cultivation of bacteria, the laboratory should be situated in a healthy locality. It should be as free as possible from contaminating influences, should be kept absolutely clean, and every aseptic precaution against the harboring or development of pathogenic organisms should be taken. To insure perfect work the greatest vigilance is needed in every department of such a laboratory.

From the moment that the milk is delivered from the farm at a temperature of about 4.4° C. (40° F.) it should be watched over and cared for with scientific accuracy during the whole process of the modification which it undergoes in the laboratory. The milk-rooms should be cool and free from dust, and isolated, so far as possible, from other parts of the laboratory.

There should also be an entirely separate room where the returned packages and all articles received from the homes of the consumer should be directly brought from the street or wagons, and where these articles can be immediately sterilized in apparatus reserved for this purpose.

The modifying materials used in the laboratory should be carefully kept for use in glass vessels, and at a temperature of about 4.4° C. (40° F.), to prevent the growth of bacteria. The reason for this is that milk modified from materials free from bacteria is not only better for the infant than milk in which the bacteria have been destroyed by heat, but that there are certain toxins which cannot be destroyed by heat, and by their virulence may cause serious disturbance even after the most thorough sterilization has been carried out.

In a carefully guarded milk-supply also it is seldom necessary to ever pasteurize a milk which is sent out from a well-equipped laboratory. On the other hand, modification has been found to fail entirely when applied to old milk, dirty milk, and to milk bacteriologically impure.

A special room should be provided for the milk-modifiers who are to put up the mixtures required by each prescription. There should either be a room in the laboratory where the milk is separated by means of machinery and where it can be tested and steamed, or this can be done in the milk-room at the farm. The office at the laboratory should be entirely separate from these work-rooms, so that customers coming to leave their orders should not go near the materials used for modification and thus possibly contaminate them. All odors should be excluded from the work-rooms, as they are absorbed by milk very quickly. It is hardly necessary to say that the employees of a laboratory, whether they be in the office or in the work-rooms, should be intelligent and interested in their work, and that the modifying clerks should be especially instructed in the necessity of absolute cleanliness, should be made to understand the dangers of sepsis and the importance of asepsis, together with a practical knowledge of how to guard against and exclude pathogenic organisms.

MILK-ROOM.—The milk should be aërated and cooled to about 6.66° C. (44° F.) at the farm, then packed in ice in such a way as to maintain its temperature during transportation below 7.22° C. (45° F.), and delivered to the milk-room within a few hours of the time of milking.

This milk, as a result of the especial manner in which the cows have been fed and cared for and the selection of them according to the proper breed, may be said to have an almost uniform percentage of its elements. Even at those times of the year when the percentages of the different elements of milk commonly vary from changes in the pasturage and in the habits and surroundings of the animals, the milk of these cows, which have their food supplied to them in stated rations at one time of the year as well as another, is not subject to the elemental variations which occur in the milk of ordinary cows.

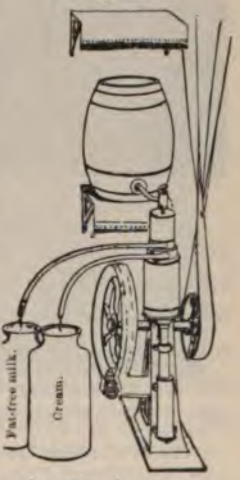
Separating-Room.—The milk can be separated either at the farm or in the laboratory in a room arranged and cared for in very much the same way as the milk-house at the farm. The walls are of white tile, and the ceilings are of material which can be washed and scrubbed. The floor is of asphalt, impenetrable to water, and is kept thoroughly moistened and free from every kind of dirt and dust.

Ventilator.—In addition to the other precautions against pathogenic germs, the air of the separating-room is kept fresh and pure by means of a ventilator. This consists of a large steel fan, which revolves at the rate of two thousand times a minute, and by the force of its current carries away any flies or particles of dust which may come within its reach. The air which comes into the separating-room, as well as into the milk-room and modifying-room, should be washed with sterile water.

Separator.—The *centrifugal separator* revolves six thousand eight hundred times in a minute, and works with such searching effect on the milk that only a small percentage (0.13) of fat remains in the separated milk.

The utility of the separator, however, does not consist wholly in its absolute withdrawal of the fat from the milk and in providing cream as fresh as to time as is the separated whole milk: it accomplishes two other very important results. First, by its great centrifugal force it separates from the cream and the separated milk any dirt or foreign matter of any kind which necessarily gets into every milk, and thus provides at once a practically clean milk, a most important result from a bacteriological point of view.

FIG. 47.



Centrifugal separator.

Still.—A still for freshly preparing each day distilled water is part of the laboratory equipment.

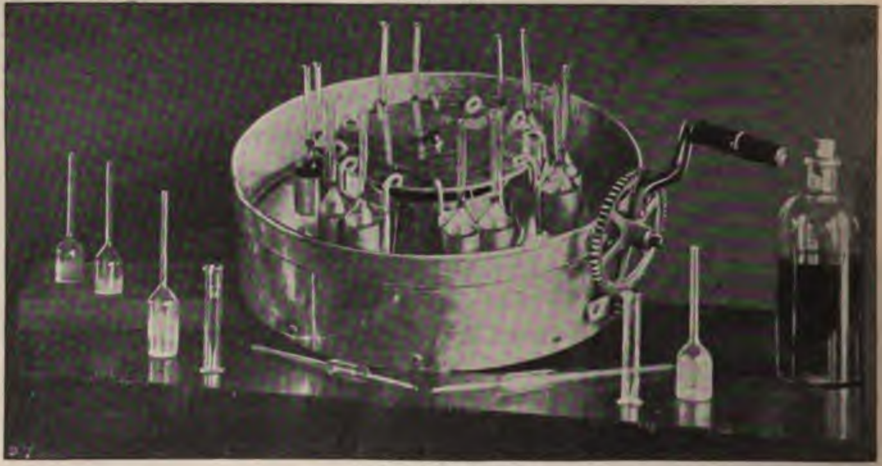
Modifying-Room.—In the modifying-room the milk is tested and the modification of the milk is completed.

Babcock Fat-Tester.—To be doubly sure that the chemistry of the milk is what we suppose it to be from the uniform nature of the primal milk-supply, we take advantage of the knowledge which we have concerning the changes most likely to take place in certain elements of the milk.

The percentage of the proteids, of the sugar, and of the mineral matter in the milk of a herd of this kind, where uniformity in the feeding is the rule, is not apt to be appreciably affected. But the percentage of the fat in individual cows differs from day to day, and thus slightly affects the percentage of the fat in the milk of the herd.

The fat, then, being the element by which we know whether each milking gives a uniform product, we test this element by means of what is called the "Babcock Fat-Tester." Fig. 48 shows the Babcock machine.

FIG. 48.



Babcock fat-tester.

To determine the percentage of fat, test-bottles containing the acidified milk are placed in a centrifugal machine, by the rapid revolution of which the fat is made to separate quickly and completely. The milk is acidified in order that the proteids, casein and fibrin, may be changed to soluble acid albumins, which offer less resistance to the rising and aggregation of the fat-globules.

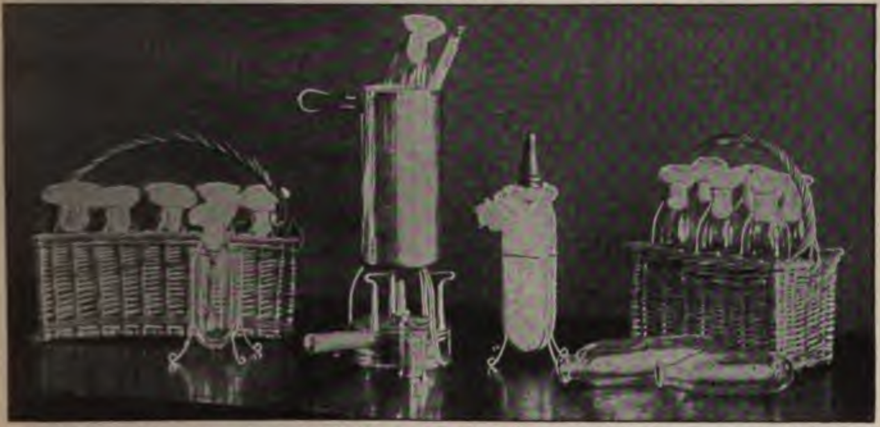
Approximately equal volumes of milk and commercial sulphuric acid of 1.82 specific gravity are mixed in a test-bottle with a long graduated neck. A pipette, delivering about 17.5 c.c. of milk, and a measuring cylinder for the acid, are used. The bottles are whirled for several minutes at a temperature of 93° C. (200° F.) in a horizontal wheel making from seven to eight hundred revolutions per minute. This wheel is surrounded by a copper jacket, which may be filled with hot water for heating during the test. The separation of fat by gravity alone is not complete even when the bottles are left standing for several hours. By the centrifuge, however, a perfect separation is accomplished in a few minutes. If whirled at once, no heat need be applied, as that caused by the strong acid and milk is sufficient. After whirling, the bottles are filled to the neck with hot water, returned to the machine, and whirled for one or two minutes longer, after which they are filled with hot water to about the seven per cent. mark, and the machine is again turned for a short time, the temperature being kept up by means of a lamp or by filling the jacket with hot water. The fat separates and its percentage is noted while still liquid, preferably at about 65° C. (150° F.), the reading giving the percentage of fat directly without calculation and being easily taken to 0.1 per cent.

This daily testing of the fat enables the modifier to preserve the accuracy of his material, and to correct any variation in the percentage of the cream as it comes from the separator.

Knowing the exact percentages contained in the cream and milk, the office clerk can, by a simple mathematical formula, give the required directions on the modifying clerk's formula for obtaining whatever percentages of the other elements the physician may call for.

Apparatus for the Transportation of Modified Milk.—Fig. 49 represents the various forms of apparatus which are provided for feeding the infant in its home.

Fig. 49.



In the left of the picture is a basket holding eight tubes of a capacity of six ounces each. In front of this basket is a four-ounce tube in a wire stand. In the middle of the picture is a tin apparatus for warming the milk at the time of feeding. An alcohol lamp is shown beneath the warmer, and a tube of milk and a thermometer for testing the temperature of the milk are in the tin warmer. Next to and to the right of the tin warmer is a tube with a capacity of eight ounces. It is enclosed in a white worsted cozy, has the rubber nipple in place, and is supported in a wire stand. In the right of the picture is a basket containing six tubes with a capacity of eight ounces each. In front of this basket are an eight-ounce tube and a four-ounce tube.

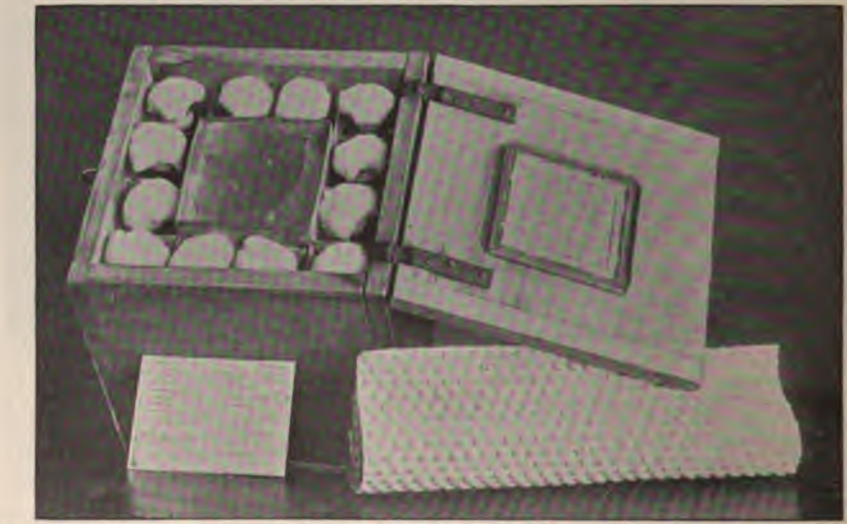
This apparatus is very simple and practical for transportation. A wicker basket, divided into a number of compartments corresponding to the number of feedings which are to be sent to the infant, has been found to be the most practical. These baskets with their tubes can be placed directly in the sterilizer, and are not harmed by the heat to which it is necessary to expose the food.

This tin receptacle can be placed above an alcohol lamp; the water in it is to be on a level with the height of the milk which is contained in the tube, and the tube is submerged in the water. It has been found necessary to take the temperature of the food by means of a thermometer placed directly in the tube. No rule can be laid down by which the temperature of the water-bath determines that of the milk, unless the tubes are of uniform thickness and the milk uniform in quantity and temperature when placed in the bath. The thermometer must be washed in sterilized water with the greatest care, both before and after it is used. The food when given to the infant should have a temperature of from 36.6° to 37.7° C. (98° to 100° F.).

As in direct feeding from the breast the food which the infant receives has the same temperature at the end of the feeding as at the beginning, we should copy this provision of nature and not allow the temperature of the food to vary during the time it is being taken. To accomplish this end, a white worsted cozy can be used. The cozy is warmed at the same time that the milk is being heated, and the tube when placed in it is prevented from cooling. Thus the infant receives a food of unvarying temperature throughout the whole of the feeding.

Fig. 50 represents an ice-box which can be used in hot weather, and has proved to be of great practical utility. It admirably serves the purposes of an express box and of a home refrigerator. The ice is packed in a metal compartment in the middle of the box, and the tubes are placed, each in its own compartment, around the sides of the ice-receptacle.

FIG. 50.



Ice-box, holding twelve tubes. Receptacle for ice in centre of box. Laboratory prescription-blank in front of box, and packing paper under end of open lid.

MATERIALS FOR THE MODIFICATION OF MILK.—The *cream* as well as the *fat-free milk* contains its own definite percentages of sugar, proteids, and mineral matter. The following analysis shows the percentages of the fat, sugar, and proteids in a laboratory sample of sixteen per cent. cream as compared with the fat-free milk:

	Fat.	Sugar.	Proteids.
Cream	16.00	4.00	3.60
Fat-free milk	0.13	4.40	4.00

To provide the means for adjusting the percentages of the *sugar* which are called for, a carefully prepared twenty per cent. solution of milk-sugar and distilled water is used. The reaction of the food is adjusted by means of *lime-water.*

In addition to the materials used for purely milk mixtures, other articles of food, such as freshly prepared oats, barley, and wheat, can be obtained at the laboratory, and these can be prescribed by the physician in any combination or according to any stated percentage of fat, sugar, proteids, or starch which he may wish. By calculation also a whole milk can be used in place of the separated milk, and a gravity cream in place of a separated cream, if a few hours' notice is given by the physician who is prescribing the mixture. It is well that this latter statement should be noted, as physicians are so apt to think that only separated milk and cream are used at the laboratories, and that the food is always sterilized or is always made alkaline with lime-water. This is not so, and it should be thoroughly understood that the laboratories are ready to provide whatever the physician orders, and in whatever way he orders, and that they are not allowed to do anything else, either to prescribe, to change the prescription, or to sell a modified milk preparation without a physician's prescription,—that is, the laboratory is merely an instrument in the hands of the physician, and is in no way responsible for the results obtained from the feeding, except so far as freshness of material and exactness of combination is concerned. The physician, on the contrary, can order his preparations to be delivered unheated or at any temperature, whether it be 65.5° C. (150° F.), 75° C. (167° F.), or 100° C. (212° F.); alkaline or not, as he pleases, and if alkaline, made so in any way he prefers to order, such as by lime-water or soda; his fat percentages can be given him with gravity cream if he prefers it to separated cream, and his proteids with whole milk instead of separated milk; also his sugar can be cane-sugar if he prefers it to milk-sugar.

The physician, however, should appreciate that separated milk contains far less dirt and much fewer bacteria than whole milk, and that using gravity cream means a six or eight hours' older cream, and necessarily more bacteria than a quickly separated cream, which gives us as fresh a material for modification as the milk. I personally have never been able to satisfy myself that the emulsion of the fat was in any way disturbed by the separator, and my clinical experience, which has been large with both separated and unseparated milk and cream, has never shown that any harm came from using the former.

With these modifying materials the modifying clerks combine each infant's food according to the prescription before them, and pour it into the glass tubes from which the infant is to nurse. These tubes, which have been especially devised as the most practical for general use, are adapted both for transportation and for use as nursing-bottles, and are easily cleansed.

There are two sets of clerks. One set is engaged in modifying the milk according to the prescriptions. As soon as the tubes are filled by the modifying clerks they are passed on to the stoppling clerks, who immediately seal them with aseptic non-absorbent cotton especially prepared for this purpose, and place them in baskets, the compartments of which are adapted to the number of feedings ordered for the special infant. The tubes are kept on tube-racks within easy reach of the modifying clerks. Each basket has its own label attached to it, with the address of the person to whom it is to be sent.

The rule of absolute cleanliness is carried out in every possible detail, from the table on which the materials are combined to the dress and hands of the clerks.

The milk is thus separated and recombined according to the prescriptions, stoppled, and placed in their respective baskets for transportation.

STERILIZATION OF THE MILK.—The sterilizer is so arranged that the steam which passes through it can be regulated so as to produce any degree of heat required up to 100° C. (212° F.). This is accomplished by a regulator attached to the steam-pipe. The man in charge of the heating of the food, by keeping his hand on the regulator and his eye on the thermometer which is fitted to the sterilizer, can subject the baskets and the tubes in them to whatever degree of heat is ordered, and of course for the length of time required.

The question whether milk should be boiled or steamed is one which is not of much significance, and can be settled according to the fancy of the individual practitioner, a greater or less destruction of the bacteria contained in the milk taking place according to the degree of heat to which it is submitted. My own experiments in comparing steamed with boiled milk show that the odor and taste of boiled milk are present when milk is steamed, but to a much less degree than in boiled milk; also that while a thick scum is formed on milk boiled for twenty minutes, which is tenacious and does not disappear on shaking, only a very thin scum forms on milk steamed for twenty minutes, and that this is not tenacious and almost entirely disappears on shaking.

After the food has been heated, the baskets are taken out of the sterilizer and placed in the cooling-tank, where the temperature of the food is reduced to 13.3° C. (38° F.). The baskets are then placed in the delivery-wagon, which conveys them to their various destinations.

When the baskets are delivered at the homes of the consumers, the baskets and tubes of the previous day are returned to the laboratory. When they reach the laboratory they are taken directly from the street to the wash-room, which is entirely shut off from the rest of the laboratory.

WASH-ROOM.—In the wash-room, in order to carry out absolutely the aseptic precautions, the baskets and everything which has been returned to the laboratory are placed in a special sterilizer connected with the wash-room. The bottles, after being sterilized, are thoroughly washed in tubs, which are especially adapted for this purpose, in a solution of soda and water. All the tags and stoppers are destroyed after sterilization. The baskets are of woven willow, and are easily kept sterile.

In this way, always guarding against possible infection of all kinds, the laboratory enables us to make use of the chemical and bacteriological knowledge which we have acquired in connection with the feeding of infants, and fulfils the requirements of that system of substitute feeding which up to the present time has proved to be the best.

Principles of Prescription Writing in Percentage Feeding.—Let it be supposed that a modified milk is to be prescribed for an infant four months old with a normal digestion and of normal weight and general development. The regular prescription blank issued by the laboratory can be used, but, of course, a milk prescription can be written as one would write for a drug.

The following is a sample prescription, in which the physician writes for a total proteid without reference to the relative proportions of caseinogen and lactalbumin:

℞	Per Cent.		
Fat	4.00	Number of feedings	7
Milk-sugar	7.00	Amount of each feeding	135 c.c. (f℥ 4½).
Total proteid	1.50	Infant's age	4 months.
(a) Caseinogen		Infant's weight	14 pounds.
(b) Lactalbumin (whey proteid)		Alkalinity	5 per cent.
		Heat at	155° F.
Ordered for			
Date		Signature.	

In regard to the question of the reaction, it can be left to the milk-modifier, as we leave to him the carrying out of other directions contained in the prescription. If the milk brought to the laboratory on the special day when we are sending our prescription has been produced from cows fed, as has been previously described, on sugar-beets, the milk may be already sufficiently alkaline for an infant's digestion when normal. If, on the contrary, the milk has its usual acid or amphoteric reaction, the milk-modifier will make it slightly alkaline, in accordance with the physician's prescription and according as the milk of the special day has a greater or less acid reaction; or, if so ordered, he will make the alkalinity correspond to five per cent., ten per cent., twenty per cent., or to any percentage desired. For this purpose lime-water should be used, as being the best material and as least likely to do harm. If, however, the infant's digestion is not normal and we wish to prescribe a precise amount of lime-water, we can do so by writing for whatever percentage we choose, as we do for the other elements of the milk. In modifying the milk which comes from the farm connected with the laboratory, as a rule, one-twentieth part of lime-water (five per cent.) is sufficient to make the reaction correspond to that of normal human milk. Table 39, page 175, shows what the percentage of lime-water should be in order to obtain a greater or less degree of alkalinity. The hydrate of lime is said to be soluble to the extent of 1 part in 778 parts of water at a temperature of 15.5° C. (60° F.). This would make one ounce of lime-water to contain rather more than 0.03 gramme ($\frac{1}{2}$ grain) of CaO_2H_2, hydrate of lime.

The milk from the farms connected with the laboratory has proved to be comparatively free from bacteria, and in most instances it is unnecessary to destroy the few bacteria which exist in it; and as it is not harmful to the infant, it need not be exposed to heat. When, however, the milk has to be transported a long distance, or when the infant has a delicate digestion or is sick, it is often better to heat the milk to 68.3° C. (155° F.). This temperature is sufficient to kill those developed bacteria which would be of any harm to the digestion of the infant, and at the same time is below 72° C. (161.6° F.), the point at which coagulation of the proteids is supposed to take place. We thus obtain a practically pure fresh milk, uncooked and sterile. We therefore write in our prescription 68.3° C. (155° F.). If the milk is to be sent an unusually long distance, if the weather is hot, or if the milk-supply has to last more than twenty-four hours, a higher degree of heating can be used, according to the wish of the prescriber. Thus, 100° C. (212° F.) is a temperature used for these purposes at the laboratory. When, again, we wish the milk to be absolutely sterilized, as may be the case when we are preparing it for an ocean voyage or for a trip across the continent, not only a high degree of heat, 100° C. (212° F.), but two or three heatings, with intervals of twenty-four hours, are necessary for this complete sterilization, and this can be called for in our prescription. The length of time during which the milk should be heated, as a rule, can be left to the judgment of the superintendent. Ten minutes is often sufficient to kill the developed bacteria and to make this especially protected milk practically sterile. Experience, however, has proved that during transportation the milk is often exposed to temperatures conducive to the further development of bacteria, and that practically the bacteriological results which we obtain in the laboratory do not entirely hold when the milk is exposed to these varied conditions of transit. As a rule, therefore, from twenty to thirty minutes is the proper time to heat mixtures of modified milk sent from the laboratory.

When the prescription is sent to the office the clerk copies it into a book, which records each day's feeding of each individual infant, and then translates the physician's prescription into such form as can be readily understood by the modifying clerks. Of course this form may vary in different parts of the world, according as the metric or the apothecary system is in use. In the work of the American laboratories, although the prescriptions are written by the physicians in the metric system, it has been found more convenient, when delivered to the patrons of the laboratory, to have the amounts expressed in ounces and drachms. The office clerk, after translating the metric percentages into ounces and drachms, copies it on to a blank called the modifying clerk's prescription.

The prescription is then placed in the hands of the modifying clerk, who combines the different elements of the prescription by means of the elemental materials which have been brought into the modifying-room from a different part of the laboratory.

Practical Limits of Laboratory Modification.—I have requested physicians to write their prescriptions within certain limits as to the percentages of the fat, sugar, and proteids, and to allow the mineral matter for the present to regulate itself. The limits which up to the present time the laboratory has found it necessary to place on the prescriptions for the milk-modifiers, and within which the modifying clerk is supposed to put up the prescriptions, are as shown in the following table:

TABLE 42.

Fat	from 0.03 to 36.00
Sugar	from 0.87 to 20.00
Proteids	from 0.22 to 4.00

There is not much doubt that in the future more and more exact results will be obtained, representing definite percentages of still wider limits. The results obtained from combining the modifying materials used by the modifying clerks have so often been proved to be practically correct, that we can assume that when we write a prescription we shall obtain in return a product which in its various elements comes within a fraction of one per cent.

The following figures show the various combinations of different percentages, which when written for can be supplied by the laboratories:

TABLE 43.

Low Fats.				
Fat	0.03	0.04	0.08	0.12–16
Sugar	2.00	3.00	4–5.00	6.00–7.00
Proteids	0.75	1.00	2.00	3.00–4.00
Low Sugars.				
Sugar	0.87	1.40	2.12	3.50–4.30
Fat	2.00	3.00	3.50	4.00
Proteids	0.75	1.00	2.00	3.00–4.00
Low Proteids.				
Proteids	0.22	0.34	0.45	0.53
Fat	2.00	3.00	4.00	4.50
Sugar	2.00	3.00	4.00–5.00	6.00–7.00

The low fats show the lowest percentage of fat which can practically be used at the laboratory, and have been combined with various possible percentages of sugar and of proteids. The low sugars show, in like manner, the lowest percentages of the sugar which can be combined with these various percentages of fat and proteids. Finally, the low proteids show the various combinations which can be obtained with the fats and sugars.

THE USE OF WHEY IN PERCENTAGE FEEDING.—Although at present our knowledge of the comparative elemental percentages of the total proteids in both human and cow's milk is inexact, yet we can at least arrive at approximate results, with much benefit to an infant with weak digestion, in our endeavor to make the relative proportion of the lactalbumin in cow's milk correspond to what is probably provided by nature for purposes of nutrition in the proteid of human milk. It is evident, on examining the analysis of the proteids of human milk, that for some good reason the caseinogen is small in amount in comparison with the lactalbumin. In cow's milk, on the contrary, the lactalbumin is small in amount in comparison with the caseinogen.

Thus, while in *human milk* the lactalbumin is about two-thirds ($\frac{2}{3}$) and the caseinogen about one-third ($\frac{1}{3}$) of the total proteids, in *cow's milk* the lactalbumin is only one-sixth ($\frac{1}{6}$) to five-sixths ($\frac{5}{6}$) caseinogen. (König.) We should, therefore, first determine the total proteid percentage and then calculate the amount of whey needed to obtain two-thirds ($\frac{2}{3}$) lactalbumin and one-third ($\frac{1}{3}$) caseinogen. As with our present knowledge this is not practicable except in comparatively low proteid percentages, we should calculate to come as near these proportions as possible.

If, therefore, we are writing a prescription which calls for a total proteid of 1 per cent., we should calculate to have 0.75 per cent. of lactalbumin and 0.25 per cent. of caseinogen. A prescription calling for fat 3 per cent., sugar 6 per cent., proteid 1 per cent., alkalinity 5 per cent., would be written as follows:

℞	Per Cent.		
Fat	3.00	Number of feedings	9.
Sugar	6.00	Amount at each feeding	75 c.c. (℥2½).
Proteids (total)	1.00	Infant's age	3 weeks.
(*a*) Lactalbumin (whey proteid)	0.75	Infant's weight	9 pounds.
(*b*) Caseinogen	0.25	Alkalinity	5 per cent.
		Heat at	155° F.

It is to be noted that although the total proteid percentage in the milk for an infant may be considerably increased, it is these higher percentages which are the most irrational in their nutritive values in the early months of infancy, if we hold to the rule that the caseinogen should be only one-third of the total proteids. This ratio of lactalbumin to caseinogen can be obtained if we are writing for a low proteid, as in the above prescription, or in a prescription calling for a total proteid percentage of 0.75, of which 0.25 per cent. shall be caseinogen and 0.50 per cent. lactalbumin. If, on the other hand, we write for a high total proteid, such as 3 per cent., the highest percentage of lactalbumin that can be obtained is 0.85, and the remaining 2.15 per cent. is caseinogen, which practically reverse our ratio, making the caseinogen over two-thirds ($\frac{2}{3}$) and the lactalbumin less than one-third ($\frac{1}{3}$).

It can be said, however, that as the infant grows older its power to digest casein becomes proportionately greater, so that in the later months of infancy, the tenth, eleventh, and twelfth, its proteolytic function has become adapted to this change in the ratio of the caseinogen and lactalbumin, so that the higher total proteids, such as 2.50, 3, 3.50, and finally 4 per cent., with the relatively high caseinogen and low lactalbumin become the proper nutritive proportion for the infant.

The point especially to be emphasized is that in the early months of life, which demand a low proteid percentage, we can by the use of whey obtain, in a modified milk, the same proportions of lactalbumin and caseinogen which we find in human breast-milk at a corresponding period of infancy. A further consideration of the use of whey will be found on page 233.

The prescriptions calling for definite proportions of casein and lactalbumin which can now be filled at the laboratory are as follows:

TABLE 44.

Fat.	Caseinogen.	Lactalbumin.	Sugar.	Fat.	Caseinogen.	Lactalbumin.	Sugar.
1.00	.25	.25	4 to 7	2.50	.25	.50	4 to 7
1.00	.25	.50	4 to 7	2.50	.25	.75	4 to 7
1.00	.25	.75	4 to 7	2.50	.50	.50	4 to 7
1.00	.50	.25	4 to 7	2.50	.50	.75	4 to 7
1.00	.50	.50	4 to 7	2.75	.25	.25	4 to 7
1.00	.50	.75	4 to 7	2.75	.25	.50	4 to 7
1.50	.25	.25	4 to 7	2.75	.25	.75	4 to 7
1.50	.25	.50	4 to 7	2.75	.50	.50	4 to 7
1.50	.25	.75	4 to 7	2.75	.50	.75	4 to 7
1.50	.50	.25	4 to 7	3.00	.25	.25	4 to 7
1.50	.50	.50	4 to 7	3.00	.25	.50	4 to 7
1.50	.50	.75	4 to 7	3.00	.25	.75	4 to 7
2.00	.25	.25	4 to 7	3.00	.50	.25	4 to 7
2.00	.25	.50	4 to 7	3.00	.50	.50	4 to 7
2.00	.25	.75	4 to 7	3.00	.50	.75	4 to 7
2.00	.50	.50	4 to 7	3.50	.25	.50	4 to 7
2.00	.50	.75	4 to 7	3.50	.25	.75	4 to 7
2.25	.25	.25	4 to 7	3.50	.50	.50	4 to 7
2.25	.25	.50	4 to 7	3.50	.50	.75	4 to 7
2.25	.25	.75	4 to 7	4.00	.25	.25	4 to 7
2.25	.50	.50	4 to 7	4.00	.25	.50	4 to 7
2.25	.50	.75	4 to 7	4.00	.25	.75	4 to 7
2.25	.75	.50	4 to 7	4.00	.50	.25	4 to 7
2.25	.75	.75	4 to 7	4.00	.50	.50	4 to 7
2.50	.25	.25	4 to 7	4.00	.50	.75	4 to 7

PEPTONIZATION OF MILK.—Peptonized milk is cow's milk with its proteids partially or entirely predigested by means of the extract of pancreas and soda. There is no doubt that the proteids of cow's milk are at times a source of trouble to the infant's digestion, and that under certain circumstances they can with great benefit be treated by predigesting them for a time, and allowing a stomach which otherwise digests well to rest and recover its entire digestive power. It is of use also where a decided idiosyncrasy of the individual precludes the digestion of these constituents of the milk. In many cases the indigestion is attributed to a lack of power to digest proteids at all, while in fact the stomach is simply rebelling against an amount of proteids above the standard percentage, or against some other constituent. It would seem that, for the average infant, this predigestion of the proteids is contrary to nature's teaching. There are certain natural functions which should be allowed to act as they do on human milk, and it seems irrational and contrary to the laws of physiology not to encourage all the functions to act naturally, instead of forestalling their action and allowing them to fall into disuse and thus to be weakened. The infant's stomach is intended to digest proteids, and not to have the proteids digested for it. Clinically, also, the use of peptonized milk supports this view, for, so far as I know, no very brilliant results have been obtained from its use, *except* when the infant's digestion has been in an abnormal condition and one which has called for some decided relief from the proteid elements of milk. Peptonized whole milk, therefore, as a food for young infants is one which consists of too large an amount of digested proteids, too little sugar, and a very large overproportion of mineral matter, while peptonization of modified milk should only be given in cases in which it has been found that various combinations of the fat and sugar with very low percentages of the proteids have proved inefficient.

When a peptonization of the proteids is prescribed, the clerk at the laboratory calculates the amount of peptonizing powder required for each feeding, and sends a number of powders corresponding to the number of feedings.

The technique for the peptonization of milk is as follows:

In a clean glass jar containing 4 ounces of cold distilled or boiled water dissolve 1 gramme (15 grains) of bicarbonate of soda and 0.25 gramme (5 grains) of pancreatine (extractum pancreatis), to which add 12 ounces of the milk. Set the jar in a vessel of water at a temperature of 41.6° C. (107° F.) for from seven to ten minutes. Cool immediately, and keep on ice until used.

To peptonize modified milk an amount of the powders should be used corresponding to the percentage of the proteids in the mixture, taking the standard of whole milk to be represented by four per cent. of the proteids.

THE USE OF CEREALS IN LABORATORY FEEDING.—When a physician orders cereals to be prepared at the laboratory, he is enabled to obtain preparations containing exact percentages of the constituents of any cereal food.

In substitute feeding, the addition to modified cow's milk of starch in various forms is frequently recommended.

This brings us to the consideration whether starch should be made a part of an infant's food. Physiologically, we know that during the first ten or twelve months of life the function of converting starch into sugar is in the process of development. It is true that a partial conversion of the starch can be performed at quite an early age, and, in exceptional cases, to a much greater extent than by the average infant. It is rational to suppose that when a function is being developed it should not be taxed with a trial of the use which will later be demanded of it. That is, a function develops more perfectly if its power is not exerted too early. With these facts before us, and simply recognizing that the best known food for infants, woman's milk, does not, under any circumstances, contain starch, I believe that starch should not form a part of the infant's food in the early months of its life.

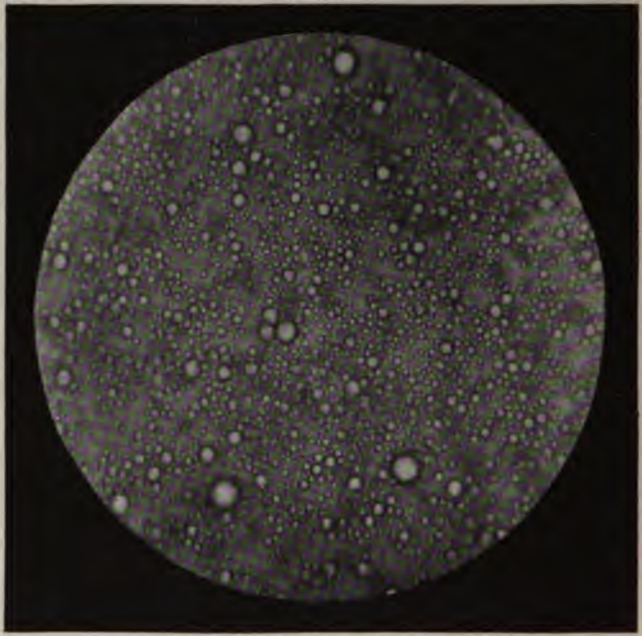
When, in the latter part of the first year, it is deemed best to give cereals, it is desirable not to give immediately a high percentage of starch in the mixture. We should not ignore the fact that the various cereals contain fat and proteids as well as starch, and that the percentage of sugar in the mixture will be raised by the converted starch. A gradual change should be made in the infant's food until its digestive capabilities have become adapted to the food values indicated for digestion and nutrition in the second year, such as are represented by a higher rate of fat, sugar, and proteids. It is well at this period to use an undiluted milk, preferably from Holstein cows, and to obtain the increased sugar ratio from the starch given plus the 4.50 per cent. of sugar in the milk. Any additional sugar that may be needed at this time may be given with the food in the form of cane-sugar, rather than milk-sugar. The starch can best be obtained from the preparations of oats, barley, and wheat, as described on page 239.

THE EMULSION IN MODIFIED MILK.—As the question has arisen in the minds of some physicians as to whether it is wise to use a centrifugal cream in making a modification, the objection being that the centrifuge destroys the emulsion, the following photomicrograph (Fig. 51) of a drop of unmodified cow's milk is interesting to study. The analysis of this especial milk was as follows:

Cow's Milk.

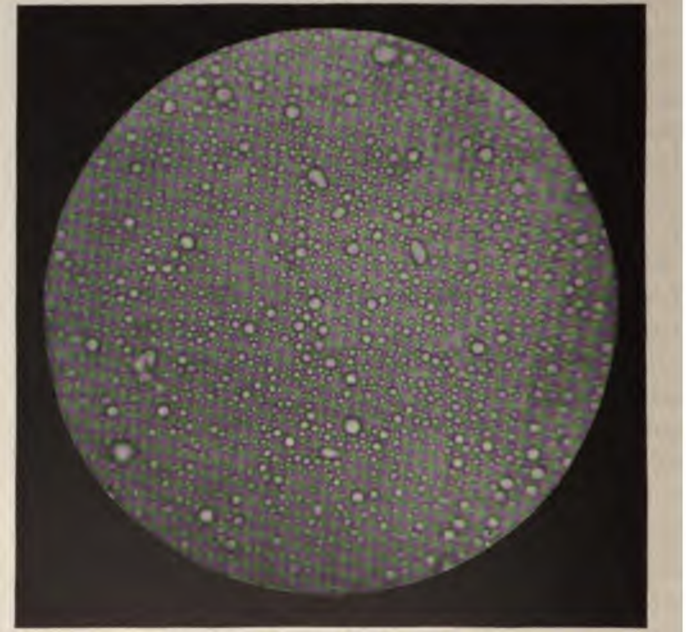
Fat	4.04
Sugar	4.55
Proteids	4.15
Mineral matter	0.71
Total solids	13.45
Water	86.55
	100.00

FIG. 51.



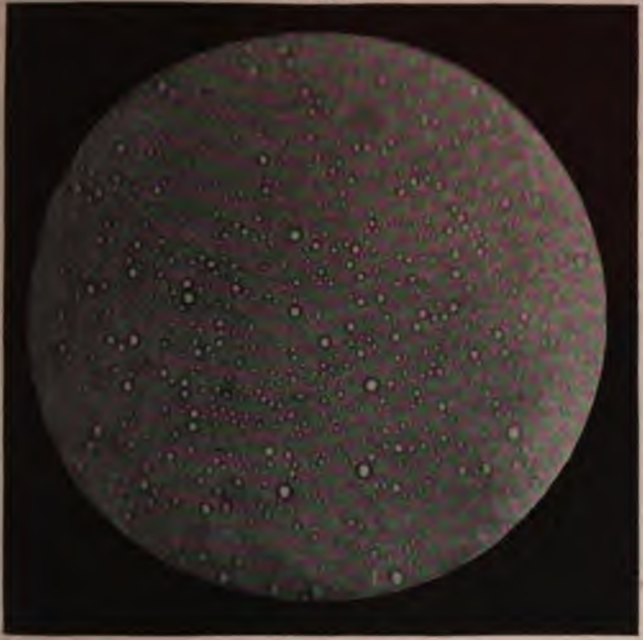
Unmodified cow's milk.

FIG. 52.



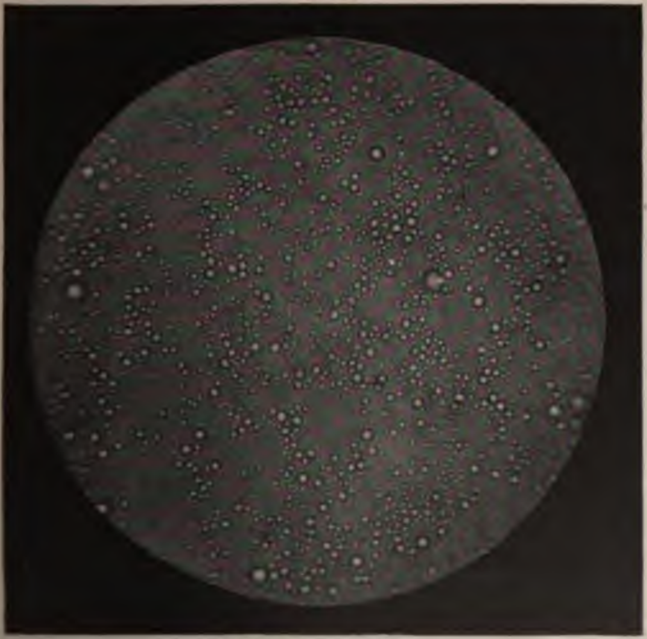
Cow's milk separated and recomposed.

FIG. 53.



Human milk.

FIG. 54.



Modified cow's milk.

Fig. 52 represents a drop of cow's milk modified to correspond to the same analysis as in Fig. 51; and in examining the two drops it will be seen that the emulsion in the modified drop is quite as fine as, if not finer than, that in the unmodified.

Fig. 53 represents a drop of human milk, and Fig. 54 a drop of cow's milk, modified so as to correspond to the percentages of the human milk, which were as follows:

Human Milk.

Fat	2.67
Sugar	6.37
Proteids	2.69
Mineral matter	0.15
Total solids	11.88
Water	88.12
	100.00

The emulsion in these two drops of milk seems to correspond very closely, and certainly does not warrant the assumption that the emulsion has been seriously interfered with by separation and recomposition.

It has been noticed that in certain instances when a cream mixture is subjected to the jar dependent upon transportation and delivery for any considerable distance, a portion of the fat has a tendency to collect on the surface of the milk in globules. Experiments made by White and Ladd under my direction have shown that transportation alone was not sufficient to produce this disturbance in the emulsion, and that it did not depend alone upon the use of centrifugal cream. Centrifugal and gravity cream mixtures, after eight to eleven hours of transportation on several successive days at a temperature such as prevails in average October weather, showed essentially the same macroscopic and microscopic appearances, and their emulsions were hardly to be distinguished from that of whole milk. Moreover, it was found possible to produce these globules in a modified milk by placing the bottles in a box, the interior of which was kept warm during transportation by means of a vessel containing hot water, which was renewed at intervals. My opinion, based on the result of these experiments, is that the globules are produced by a combination of motion of transit with heat, such as prevails in summer; and I have not found the nutritive value of the milk to be at all interfered with in the exceptional cases in which the change occurs.

ILLUSTRATIVE CASES OF PERCENTAGE FEEDING.—The following cases were fed under my direction at the milk-laboratory during the first year of their lives, and merely illustrate the changes that would naturally be made in the food during this period in the life of a healthy infant.

The first case was a male, born November 18, 1892. The table shows the record of its weight and food during its first year:

TABLE 45.

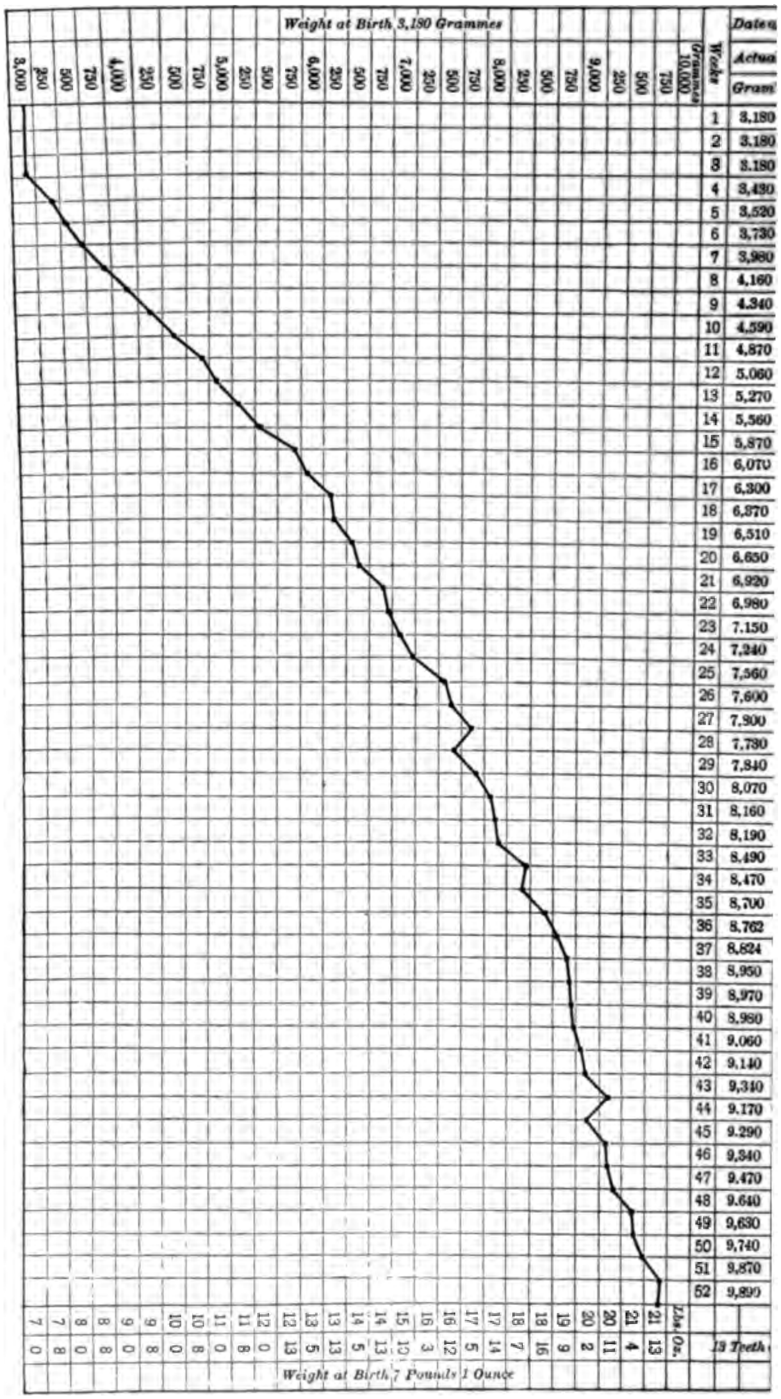
Showing Management of the Food and Increase in Weight of a Healthy Infant during the First Fifty-Two Weeks of its Life.

Date.	Weeks of Life.	Weight.			Amount at each Feeding.		Percentages of Food.				
		Grams.	Lbs.	Oz.	C.c.	Oz.	Fat.	Sugar.	Proteids.	Lime-Water.	
November 18.....	1	3752	8	6	30	1	2.00	6.00	1.00	5.00	Food heated throughout the year at 75° C. (167° F.).
	2										
	3	......	....	...	....	....	3.00	6.00	1.00		
	4	......	....	...	45	1½					
	5	......	....	...	75	2½	4.00	7.00	1.00	10.00	
December 23.....	6	4284	9	9	90	3					
	7										
	8	......	....	...	....	....	....	......	......	5.00	
January 13......	9	6944	15	8	105	3½					
	10										
	11										
	12										
	13	......	....	...	120	4					
February 17.....	14	6048	13	7	135	4½	4.00	7.00	2.00		
	15										
	16										
	17										
March 17.......	18	6748	15	1	150	5					
	19										
	20										
	21										
	22										
April 21.........	23	7308	16	5	165	5½					
	24										
	25										
	26										
May 18.........	27	7504	16	12	180	6	4.00	7.00	2.50		
	28										
	29										
	30										
	31										
June 22.........	32	7840	17	8							
	33	......	....	...	210	7					
	34	......	....	...	....	....	4.00	7.00	3.00	12.50	
	35										
	36										
	37	......	....	...	....	....	4.00	7.00	2.50		
	38	......	....	...	....	....	3.50	6.50	1.50	10.00	
	39	......	....	...	180	6	4.00	7.00	2.00	5.00	
August 17.......	40	8820	19	11	225	7½					
	41	......	....	..	....	....	....	......	......	10.00	
	42	......	....	...	....	....	....	......	......	12.50	
	43	......	....	...	....	....	4.00	6.00	2.50	10.00	
	44	......	....	...	....	....	4.00	5.00	3.00	5.00	
	45										
	46										
	47	......	....	...	....	....	Whole milk.				
	48										
	49	......	....	...	....	....	Whole milk and oat-jelly.				
	50										
	51										
November 9.....	52	9870	22								

The grammes in the third column have been reduced to pounds and ounces on the basis of twenty-eight grammes to the ounce, and the fractions of the ounce have been disregarded.

The next case was a female, born November 1, 1892. shows the line of growth in its weight from birth to the fifty- of its life :

CHART 2.



Weeks	Date Actua Gram
1	3,180
2	3.180
3	3.180
4	3,430
5	3,520
6	3,730
7	3,980
8	4,160
9	4.340
10	4,590
11	4,870
12	5.060
13	5,270
14	5,560
15	5,870
16	6,070
17	6,300
18	6,370
19	6,510
20	6.650
21	6,920
22	6.980
23	7.150
24	7,240
25	7,560
26	7,600
27	7,900
28	7,730
29	7,840
30	8,070
31	8,160
32	8,190
33	8.490
34	8,470
35	8,700
36	8,762
37	8,824
38	8,950
39	8,970
40	8.980
41	9.060
42	9.140
43	9,340
44	9.170
45	9.290
46	9,340
47	9.470
48	9.640
49	9,630
50	9,740
51	9,870
52	9,890

The following table records the quantity and quality of this infant's food during the first year:

TABLE 46.

Showing Management of the Food and Increase of Weight of a Healthy Infant during the First Fifty-Two Weeks of its Life.

DATE.	Weeks of Life.	WEIGHT.			Amount at each Feeding.		PERCENTAGES OF FOOD.				
		Grams.	Lbs.	Oz.	C.c.	Oz.	Fat.	Sugar.	Proteids.	Lime-Water.	
November 1	1	3180	7	1							Food heated throughout the year at 75° C. (167° F.).
November 8	2	3180	7	1	60	2	2.00	5.00	1.00	5.00	
November 15	3	3180	7	1	60	2	4.00	7.00	1.00		
November 22	4	3430	7	10	90	3	4.00	7.00	1.00		
November 29	5	3520	7	14	75	2½	3.00	7.00	1.00		
December 6	6	3730	8	5	....	....	3.00	6.00	1.00		
December 13	7	3980	8	14	....	....	3.00	7.00	1.00		
December 20	8	4160	9	4	90	3	4.00	7.00	1.00	10.00	
December 27	9	4340	9	10	105	3½	4.00	7.00	1.25	5.00	
January 3	10	4590	10	4	....	....	4.00	7.00	1.50		
January 10	11	4870	10	14							
January 17	12	5060	11	4							
January 24	13	5270	11	12							
January 31	14	5560	12	6							
February 7	15	5870	13	1	120	4	....	......	......	12.50	
February 14	16	6070	13	8	135	4½	....	......	......	10.00	
February 21	17	6300	14	1	....	....	....	......	......	5.00	
February 28	18	6370	14	4							
March 7	19	6510	14	8							
March 14	20	6650	14	13	150	5	4.00	7.00	2.00		
March 21	21	6920	15	7							
March 28	22	6980	15	9							
April 4	23	7150	15	15							
April 11	24	7240	16	2							
April 18	25	7560	16	14							
April 25	26	7600	16	15							
May 2	27	7800	17	6							
May 9	28	7730	17	4							
May 16	29	7840	17	8							
May 23	30	8070	18	0	180	6					
May 30	31	8160	18	3							
June 6	32	8190	18	4							
June 13	33	8490	18	15	195	6½	4.00	7.00	2.50		
June 20	34	8470	18	14							
June 27	35	8700	19	6							
July 4	36	8762	19	8							
July 11	37	8824	19	11							
July 18	38	8950	19	14							
July 25	39	8970	20	0							
August 1	40	8980	20	0							
August 8	41	9060	20	3							
August 15	42	9140	20	6							
August 22	43	9340	20	13							
August 29	44	9170	20	7							
September 5	45	9290	20	11							
September 12	46	9340	20	13							
September 19	47	9470	21	2							
September 26	48	9640	21	9							
October 3	49	9630	21	7							
October 10	50	9740	21	10	....	....	4.00	6.00	3.00		
October 17	51	9870	22	0	....	....	Whole milk.				
October 24	52	9890	22	1	....	....	Whole milk and			oat-jelly.	

The grammes in the third column have been reduced to pounds and ounces on the basis of twenty-eight grammes to the ounce, and the fractions of the ounce have been disregarded.

The following cases have a practical bearing on the method of substitute feeding by means of milk laboratories.

The first illustrates how important it is to be able to vary the percentages of the different elements of the milk, and to know that we are obtaining these variations exactly as they are ordered.

An infant was being nursed by its mother, who was healthy, and who had an abundance of breast-milk. Their summer home was by the sea-side, in a healthy situation, and the infant was surrounded with everything that could be desired for perfect hygiene. The infant during the first two months of its life nursed well, thrived, and was perfectly quiescent in its daily life. When it was three months old, the mother was very much worried by some family matters and did not take much exercise. The infant now began to have colic, and, although it gained in weight, it was very restless and cried continuously. An analysis of the mother's milk at this time gave the following result:

Fat	2.69
Sugar	6.15
Proteids	3.71
Mineral matter	0.17
Total solids	12.72
Water	87.28
	100.00

The indications for treatment were, of course, to lessen the amount of mental disturbance in the mother and to make her exercise more. The mother having followed these directions, the symptoms in the infant soon became less severe. After a few days, however, the unfavorable symptoms returned, and it was found that the mother had not been exercising and was again mentally disturbed. As it seemed impossible to regulate the function of the mammary gland under these circumstances, it was decided to feed the infant from the milk-laboratory. The following prescription was ordered:

PRESCRIPTION 3.

℞ Fat	3.50
Sugar	6.50
Proteids	1.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).
Eight tubes, each holding	90 c.c. (3 ounces).

The infant digested this food perfectly, had no colic, and again became tranquil. As, however, it only made a slight gain in weight during the first two or three weeks of this substitute feeding I changed the prescription to the following one:

PRESCRIPTION 4.

℞ Fat	4.00
Sugar	7.00
Proteids	1.50

On taking this food the infant began to make regular gains in weight, and continued to thrive until it was four months old, when it was brought back to its city home, where it was subjected to many of the annoyances which are so frequently seen, and which, although somewhat disastrous to the infant, tend to advance our knowledge of substitute feeding. The infant was surrounded with too much excitement, and was exposed to unnecessary changes of temperature in its home. During the process of removal from the sea-side to the city it caught a slight cold, and had intestinal symptoms characterized by loose discharges from the bowels and undigested food. This condition was easily obviated in a few days by simply changing the prescription at the laboratory to the following one:

PRESCRIPTION 5.

℞ Fat	2.50
Sugar	5.50
Proteids	1.00
Lime-water	10.00

Under this treatment the food was again fairly well digested, the discharges lessened in frequency, and were of a better character. The infant, however, during this sickness had lost over 224 grammes (about ½ pound) in weight.

At this juncture the grandmother of the infant so influenced the mother that she insisted upon having a wet-nurse procured at once. Although I did not approve of this change, the family were so urgent in their demands for a wet-nurse that I procured one for them. This wet-nurse was nursing her own infant and another infant at the Infants' Hospital. Both infants were thriving in every way. An analysis of this wet-nurse's milk gave the following results:

Fat	2.92
Sugar	6.20
Proteids	4.62
Mineral matter	0.16
Total solids	13.90
Water	86.10
	100.00

The milk for this analysis was taken from the middle of the nursing. The percentage of proteids was so high that I did not dare to allow the foster-infant to be put to the breast at once. I therefore endeavored to regulate the percentages of the elements of the wet-nurse's milk in the usual way. At the end of two days another analysis of her milk was made, with the following result:

Fat	3.39
Sugar	5.95
Proteids	4.78
Mineral matter	0.21
Total solids	14.33
Water	85.67
	100.00

The extraordinarily high percentage of proteids in this analysis made me absolutely refuse to allow the foster-infant to begin with its nursing from the wet-nurse. The family, however, were very impatient, and argued that, as the other two infants were gaining in weight, digesting well, and looking remarkably ruddy, it must be a good milk which they were receiving from the wet-nurse.

Two days later, although the foster-infant was decidedly improving on the substitute food from the laboratory, it happened to lose 30 grammes (about 1 ounce) in weight, and the family then insisted that this wet-nurse should be tried. Another analysis of the wet-nurse's milk was then made, and showed that the percentage of the proteids had been reduced to between 3 and 4.

I had already endeavored to find other wet-nurses whose milk would better correspond to what the infant needed, but was unsuccessful in obtaining any the analysis of whose milk showed the percentage of the proteids to be below 3.

The analyses of the milk of two of these wet-nurses were as follows :

Fat	3.88
Sugar	6.55
Proteids	3.14
Mineral matter	0.14
Total solids	13.71
Water	86.29
	100.00

Fat	3.39
Sugar	4.50
Proteids	4.70
Mineral matter	0.18
Total solids	12.77
Water	87.23
	100.00

The first wet-nurse was then brought to the foster-infant's home, and the infant was put to the breast. It absolutely refused to take the breast for twelve hours, although it was crying with hunger. Finally it was induced to nurse, but immediately after the nursing had an attack of colic. These attacks of colic were moderately severe, and occurred after each nursing. The infant soon appeared to like the milk and took it eagerly at the regular nursing intervals. In twenty-four hours from the time when the infant began to nurse its bowels were again affected. The number of discharges became frequent, and the milk evidently was not being digested well. These conditions lasted for several days, when it was found that the infant had lost over 480 grammes (about 1 pound) in weight. As the severity of the colic was increasing, and as the infant had lost its color, the mother agreed to have the feeding by the wet-nurse discontinued. I then wrote the following prescription to be put up at the laboratory :

PRESCRIPTION 6.

℞ Fat	2.00
Sugar	5.00
Proteids	1.00
Lime-water	10.00
To be heated to	75° C. (167° F.).

This mixture was given to the infant. In twenty-four hours the number of discharges from the bowels grew less, and in a few days became almost normal. It began to gain in weight, and though seeming very hungry, looked better and ceased to have colic.

The prescription was then changed to the following one :

PRESCRIPTION 7.

℞ Fat	3.00
Sugar	6.00
Proteids	1.00
Lime-water	5.00

On taking this food the infant began to make regular gains in weight, but still seemed hungry, so that at the end of another week the prescription was changed to the following one :

Prescription 8.

℞	Fat	4.00
	Sugar	7.00
	Proteids	1.50

The infant now improved steadily. It made the normal average daily gains in weight, and soon recovered its color and former strength. From this time it continued to thrive.

This case is interésting in many ways. It was very evident that a percentage of proteids over 3 was more than this especial infant could digest. It therefore had to be weaned from its mother. The wet-nurse's milk, which was agreeing perfectly with her own infant and with another infant which she was nursing at the hospital, had a percentage of proteids between 3 and 4. As I knew from my experience with the mother's milk that this high percentage of proteids would not agree with the infant, I was not surprised to find that, instead of agreeing with it, it made it sick. This case substantiates the statement that, while there are many varieties of good milk, there are also many infants who cannot thrive on them all, but only upon such as suit their individual digestive powers.

It is interesting also to record in this case that as the infant grew older, it was found that the percentage of the proteids could be increased in its food without harming its digestion, and that by the time it was eight months old it was having in its food percentages of proteids between 3 and 4, the very percentages which caused such serious digestive disturbance when it was younger. When it was ten months old it was able to digest 4 per cent. of proteids in its food.

This case as a whole so well illustrates the use of the milk-laboratory that it is hardly worth while to multiply instances of its value. I believe that by the establishment of these laboratories a new era has been entered upon in the province of infant feeding, and one which will enable us to produce results which have never before been obtained.[1]

The following prescriptions which I have sent to the laboratory at different times will give you a very fair idea of the simplicity and precision of substitute feeding.

Prescription 9.

A girl 6 years old; duodenal jaundice (functional).

℞	Fat	0.50
	Milk-sugar	6.00
	Proteids	3.00
	Lime-water	10.00

12 tubes, each 4 ounces.

---

[1] The first laboratory for the exact modification of milk that has been established in the world was opened to the public in 1891 in Boston, under the name of the Walker-Gordon Laboratory. The development of the system of modification in percentages and the establishment of this as well as of eighteen other laboratories in different parts of the country and in London, which now represent the Walker-Gordon Company, was accomplished by the joint efforts of Mr. G. E. Gordon, Mr. G. H. Walker, and Mr. J. H. Waterhouse.

PRESCRIPTION 10.

A boy 6 weeks old; healthy.

℞ Fat	3.00
Milk-sugar	7.00
Proteids	1.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

12 tubes, each 2 ounces.

PRESCRIPTION 11.

A boy 6 months old; healthy.

℞ Fat	4.00
Sugar	7.00
Proteids	2.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

8 tubes, each 6 ounces.

PRESCRIPTION 12.

A girl 4 months old; proteid digestion weak.

℞ Fat	4.00
Sugar	7.00
Proteids	0.75
Lime-water	5.00
Heated to	75° C. (167° F.).

8 tubes, each 4 ounces.

PRESCRIPTION 13.

A boy 6 months old; sugar digestion weak.

℞ Fat	3.00
Sugar	4.00
Proteids	2.00
Lime-water	5.00
Heated to	75° C. (167° F.).

8 tubes, each 6 ounces.

PRESCRIPTION 14.

A girl 4 months old; summer diarrhœa. Food has to be sent to a distant town by express.

℞ Fat	2.50
Sugar	6.00
Proteids	1.00
At time of each feeding add lime-water	½ ounce.
Heated to	100° C. (212° F.).

20 tubes, each 3½ ounces.

In this case the diarrhœa had not been sufficiently studied to determine whether it was putrefactive or fermentative, so that a safe general prescription was sent to begin with. The lime-water had to be introduced at each feeding on account of the 100° C. (212° F.) heating, necessitated by the hot weather and the distance to be sent. If the lime-water had been introduced at the laboratory and heated to 100° C. (212° F.) with the food, a reaction would have taken place between the lime and the sugar, and the mixture would have turned brown and have had a peculiar taste.

FEEDING OF AVERAGE INFANTS BORN AT TERM.—When an infant is born at term, is of normal development and weight, and is healthy, I am in the habit of regulating the quantity of its food according to the table on page

188. The quality of the food with which I usually begin is as shown in the following prescriptions. These percentages, however, are intended only to be provisional until the proper amount for the individual has been ascertained. When these prescriptions are used the infant is supposed to be digesting well and gaining in weight progressively.

PRESCRIPTION 15.

For the first twenty-four to thirty-six hours of life.

℞ **Milk sugar, five-per-cent. solution, in sterilized distilled water.**

PRESCRIPTION 16.

First week.

℞ **Fat**	2.00
Sugar	5.00
Proteids	0.75 (or .25 or .50).
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION 17.

Second week.

Fat	2.50
Sugar	6.00
Proteids	1.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION 18.

Third week.

℞ Fat	3.00
Sugar	6.00
Proteids	1.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION 19.

Four to six weeks.

℞ Fat	3.50
Sugar	6.50
Proteids	1.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION 20.

Six to eight weeks.

℞ Fat	3.50
Sugar	6.50
Proteids	1.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

PRESCRIPTION 21.

Two to four months.

℞ Fat	4.00
Sugar	7.00
Proteids	1.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription 22.

Four to eight months.

℞ Fat	4.00
Sugar	7.00
Proteids	2.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription 23.

Eight to nine months.

℞ Fat	4.00
Sugar	7.00
Proteids	2.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription 24.

Nine to ten months.

℞ Fat	4.00
Sugar	7.00
Proteids	3.00
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription 25.

Ten to ten and one-half months.

℞ Fat	4.00
Sugar	5.00
Proteids	3.25
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription 26.

Ten and one-half to eleven months.

℞ Fat	4.00
Sugar	4.50
Proteids	3.50
Reaction	Slightly alkaline.
Heated to	75° C. (167° F.).

Prescription 27.

Eleven to eleven and one-half months.

℞ Unmodified cow's milk.

At about the tenth or eleventh month I usually begin to give at first one and then two meals daily of equal parts of oat-jelly, with plain cow's milk heated to 68.3° C. (155° F.), and a little salt added according to the infant's taste at the time of the feeding. Freshly prepared barley or wheat can, if preferred, be given with milk at this age.

In the twelfth month I usually accustom the infant to taking a little bread one day old with its milk, and to be fed from a spoon, so that by the time it is a year old it is taking bread and milk for its breakfast and supper, and oat-jelly and milk for the three middle meals.

Influence of Fat on the Color of Fæcal Dejections.—I have considered it of some scientific and practical interest to record the color of the fæcal discharges which corresponds apparently to the percentage of fat in human milk and in the corresponding modified milk. Plate III., 3 and 4, facing p. 84, show the color of the normal yellow dejections of two infants who were being nursed by their healthy mothers and were themselves digesting well and thriving.

Plate III., 8 and 9, facing p. 84, represent the color of the normal yellow dejections of two infants who were fed on a modified milk which corresponded to average human milk. The percentages of the fats, sugar, and proteids in this modified milk were respectively 4, 7, and 1. The infants were digesting well and thriving.

The resemblance in color and consistency between the fæcal discharges resulting from human milk and from modified milk in which the percentage of fat was 4 is very striking.

Plate III., 7, facing p. 84, shows the fæcal discharges of a healthy infant fed on a modified milk having a percentage of 3 for its fat, 6 for its sugar, and 1 for its proteids. The color of the yellow is much lighter.

This change of color is still more strikingly illustrated in Plate III., 6, facing p. 84, in which the infant was taking modified milk composed of fat 2 per cent., sugar 5 per cent., and proteids 1 per cent., and in which the resulting fæcal discharge had a very much lighter color than in the other cases.

HOME MODIFICATION.—In arranging an infant's food it is best to adopt the most simple, precise, and scientific means of expressing what we wish to prescribe. This is done by thinking, speaking, and writing our prescriptions in percentages and by having the food put up by trained clerks in a milk-laboratory. In this way we are more certain of obtaining what we prescribe than by any other method. When, however, a milk-laboratory is not accessible, or if for any reason, such as that of expense, laboratory feeding is not available, the mother or nurse should be taught to modify the milk in the infant's home. This is what is called *Home Modification*, and under these circumstances much must be taken into consideration regarding the details of obtaining and using those materials for the modification of milk which it is unnecessary to know when the same modification is ordered at a milk-laboratory. It is, in fact, these very details which the laboratory, as a saver of time and an instrument of precision, supplies for us, and thus makes the exact knowledge of them unnecessary.

Difficulties and Dangers of Home Modification.—It must be impressed upon the minds of those who are attempting to modify milk that what they are using for this purpose may be a source of great danger to the infant consumer. Milk is a dangerous food unless it is carefully protected, so that it is first necessary to explain wherein the danger lies, and then to learn how to avoid it. Thus only can milk and cream be safely used for infant feeding in a household.

One of the chief dangers from milk and cream lies in the fact that they are such good culture-grounds for pathogenic organisms. The germs of such diseases as diphtheria, scarlet fever, typhoid fever, and tuberculosis are well known to be transmitted by milk, and, in addition to this, with very little exposure, milk becomes filled with various forms of bacteria, many of them virulent, such as the toxin of cholera infantum, and all of them foreign to the original purity of the milk as it is elaborated in the mammary gland. In this way not only may the diseases just referred to be produced, but also various forms of fermental diarrhœa and of ileo-colitis.

The most simple way to avoid the dangers arising from pathogenic organisms and from using milks and creams of unknown percentages is to obtain these milks and creams of known percentages for modification from the milk-laboratories in sealed glass jars. When this is not possible, certain primal precautions are to be taken. As the milk-ducts of the cow contain, especially in their lower parts, numerous bacteria which have gained entrance from without, the ducts should be freed from these bacteria by milking a few ounces from each teat, so as to get a milk as free from bacteria as possible. Other common sources of contamination, such as uncleanness of the cow herself and her udders should also be avoided. The cow should be milked in a clean place and by milkmen with fresh, clean clothes, and whose hair and hands have been thoroughly washed and dried with fresh, clean towels. Disease of a virulent nature has been proved to have been transmitted through the milk with disastrous effect to the consumer by the diseased finger of a milker.

Another and by no means trivial source of danger is the giving of improper percentages of the different food-stuffs to the infant in improper combinations. This may arise not only from the ignorance of the prescriber as to what combination of percentages ought to be given in the especial case, but also because he may be calculating his percentages on a basis which does not correspond to the percentages of the especial materials which he is using, materials which must necessarily vary from day to day. Under these circumstances the infant may not only suffer from all grades of indigestion, leading to serious gastro-enteric disturbance, but also contract such diseases of nutrition as infantile atrophy, rhachitis, and scorbutus without the prescribing physician knowing on what food-combinations these diseases have been contracted, for the simple reason that, while he has prescribed on paper what he supposes to be the correct food for the especial case, an entirely different food may result from his not taking into account in his calculations the great variety of percentages with which he is dealing, and which may arise not only from his failure to appreciate that slight changes in the quantities of the materials he is using may result in great percentage differences in the food, but also from the fact that although he may calculate the quantity of each material correctly, his calculations are rendered worthless by the numerical variation in his factors from day to day. These differences in percentages have over and over again in delicate infants proved to be of such serious consequence that they cannot be ignored, and every known means should be employed to avoid them; that is, the physician should know definitely what combination of percentages he is giving in the especial case, and then if this combination does not suit the case he will at least know that it is his own misconception of what was best to give, and not the fault of a combination of percentages, which was really the correct one, but which through his mistake was never really given to the infant.

The difficulty of obtaining a mixture of definite percentage in a home modification, in which a milk of unknown fat percentage is used may be emphasized in the following manner. Let us suppose that a home modification calling for a 2 per cent. fat is made. If the milk in this case is from a Jersey cow with high percentage of fat, as, for example, five per cent., the resulting proteid will be 1.60 per cent. On the other hand, if the milk used to obtain this same 2 per cent. of fat in the modification is from a Holstein cow yielding a 3 per cent. fat in the milk, the resulting proteid would be (the same quantity of materials being used) 2.66 per cent. Therefore, unless the physician knows the fat percentage of the milk he is using, he may, by means of the same formula for modification, have a variation in his proteids of from 1.60 per cent. to 2.66 per cent.

Another illustration of what different results in the percentage of the proteids may arise from using creams of various percentages is shown in the following table:

TABLE 47.

Cream used in the modification.	Fat obtained by calculation in the mixture. Per Cent.	Lowest possible proteids. Per Cent.
Ten per cent.	2	0.75
Ten per cent.	3	1.13
Ten per cent.	4	1.50
Twenty-four per cent.	2	0.31
Twenty-four per cent.	3	0.47
Twenty-four per cent.	4	0.63

Here it is shown that in a given home modification unless the cream used is of known definite percentage the resulting mixture may show proteids varying from 0.31 per cent. to 1.50 per cent., a difference which in most cases of difficult digestion is of very serious import.

There are other difficulties which arise in using creams in which the percentage is exactly known. For instance, when creams, of certain percentages are used it is impossible to obtain in the modification certain percentages of the proteids, so that it is necessary in changing from day to day the proteid percentage in a modified milk to have at command creams of different fat percentages, such 10, 12, 16, or 20 per cent. Thus, if a cream with a 10 per cent. of fat is used the following are the limitations as to the lowest possible percentages of the proteids which can be obtained from it when different mixtures of fat are desired.

TABLE 48.

	Per Cent.
In a mixture calling for 2 per cent. fat the lowest possible proteid is	0.75
In a mixture calling for 3 per cent. fat the lowest possible proteid is	1.13
In a mixture calling for 4 per cent. fat the lowest possible proteid is	1.50
In a mixture calling for 4.50 per cent. fat the lowest possible proteid is	1.82

Therefore, as is often the case when we wish to use a 3 per cent. or 4 per cent. fat combined with a fractional percentage of the proteids, or at least as low as 1 per cent., it will not be possible to make this modification with a 10 per cent. cream. In like manner, with a 12 per cent. cream, it is impossible to obtain as low a proteid percentage as 1 with as high a fat percentage as 2, as is shown in the following table:

TABLE 49.

	Per Cent.
In a mixture calling for 2 per cent. fat the lowest possible proteid is	0.63
In a mixture calling for 3 per cent. fat the lowest possible proteid is	0.94
In a mixture calling for 4 per cent. fat the lowest possible proteid is	1.25
In a mixture calling for 4.50 per cent. fat the lowest possible proteid is	1.41

However difficult it may be to obtain a variety of percentage combinations when we know definitely the percentage of the creams which we have at our command, still greater difficulties arise when we have to use a gravity cream in a home modification, for gravity cream depends for its percentage of fat on the quality of the milk from which it is obtained, on the number of the hours of the setting, and on other conditions such as temperature and handling. When set for five or six hours it is rarely that a milk will give a higher than an 8 or 10 per cent. cream; if allowed to set for a longer time so much greater is the danger of contamination.

A gravity cream varies very readily from 8 to 12 per cent. so that inaccuracies cannot fail to occur and must be allowed for when a home modification of milk is made with a gravity cream. For instance, we may suppose that we wish to prescribe a milk modification demanding 3 per cent. fat, 6 per cent. sugar, and 2 per cent. proteids, and we make use of a cream with a supposed fat percentage of 10. If this cream should vary so that its fat was 8 we should have a mixture containing only 2.4 per cent. fat instead of 3. On the other hand, if the percentage of fat in the cream was 12, on using the same formula we should obtain a mixture containing 3.6 per cent. fat in place of 3 per cent. Again, if the cream had been tested and found to contain 8 per cent. fat instead of 10 per cent., and a sufficient quantity of this cream was used to produce 3 per cent. fat in the mixture, the proteids would be 2.50 per cent. instead of the 2 per cent. prescribed. On the other hand, if the cream was known to be 12 per cent. fat, and a less quantity was added, so that the fat in the mixture should be 3 per cent., the proteids would necessarily be reduced to 1.66 per cent.

Not only do decided variations in the fat percentage of a gravity cream arise from the milk of one cow or herd, but there may also be a decided variation in the creams obtained under the same conditions but from different herds, and these variations occur whether the cream is siphoned, poured off, or skimmed.

In order to obtain a definite idea of the variation in the fat percentage of milk such as one would be likely to purchase for the purpose of home modification, I had a series of observations made upon the milk obtained from five different dairies in Boston, and hence, presumably, from as many different herds. The milk was purchased for three successive days and subjected to the same conditions, and on each day each sample of milk yielded practically the same results. In each instance the milk was allowed to set for eight hours at a temperature of about 3.3° C. (38° F.), and the top quarter carefully poured off and the strength of the resulting cream tested with the following results:

TABLE 50.

	Per Cent.
Herd A gave a cream with a fat percentage of	6.2
Herd B gave a cream with a fat percentage of	7
Herd C gave a cream with a fat percentage of	10
Herd D gave a cream with a fat percentage of	11
Herd E gave a cream with a fat percentage of	12

These experiments seem to show conclusively that given a milk of unknown strength, the top quarter may vary in its fat percentage from 6 per cent. to 12 per cent., and one might obtain very wide differences in the percentages of a home modification, in which the fat percentage of the milk used is not estimated.

Variations are especially liable to occur when the milk of one or two cows or of a small herd is used, for it is not unusual to find that certain cows give widely different cream percentages in their milk, such as from 8 per cent. to 14 per cent., thus making a possible variation of 6 per cent., and it has also been shown by the records of the experiment station at Durham, New Hampshire, that out of one hundred herds the percentage of fat in the mixed milk of each herd varied from 3 per cent. to 6 per cent.

It is therefore important for the correct home modification of milk that the physician should know the analysis of the milk of the special herd of cows which he is using, to see whether all his patients are being fed from that herd or from a number of herds, and to determine the percentage of fat in the cream from the same herd on different days.

The variation in the fat percentage in different herds is apt to be considerable, as the owner often has a preference for one or another kind of stock, and frequently has a larger portion of that particular kind in his herd. It is also well known that the milk of the whole herd, for reasons of convenience, is not mixed together each day, and that, therefore, the testing of such milk on certain days is misleading, as it may contain little or much fat, according as it may have been produced by the poorer or richer milk producers of the herd.

Owing to the different conditions of the cows and of the feeding during the year, and to the fact that the fresh cows which are giving the larger quantities of milk will at times happen to be cows giving a low per cent. of fat, while at other times the large milkers will be cows which yield a high per cent., the variation in the percentage of fat from the same herd may in the course of the year often vary as much as 2 per cent. It should, therefore, be appreciated that unless the cows are especially cared for, as are the herds connected with the milk-laboratories, the percentage of fat is constantly varying. If, therefore, it is necessary for the physician to use different herds in prescribing, it will be necessary for him to use a different formula for each herd; or if he should use the same herd, he will be obliged, in order to get the same percentage, to use a different formula at different periods.

These statements hold true when the milk can be obtained at once after milking and under the most favorable circumstances. If, on the contrary, an ordinary milk is set after it has passed through the hands of a city milkman, the same rules will not apply for obtaining, for instance, a 10 per cent. cream, as the cream will be likely to have partly risen when the milk is delivered, and if it is then set for eight hours, a twenty- or twenty-four-hour cream may be the result, which is not only objectionable because the cream is old, but because it varies greatly in its fat percentage.

In making a home modification it must also be noted how appreciably the total amounts of milk and cream used for a mixture in which specified percentages of fat and proteids are desired must differ according to the cream used. For instance, in a 40-ounce mixture, in which 3 per cent. fat and 1.50 per cent. proteids are prescribed, 8.1 ounces of milk and 7.3 ounces of cream will be required if the cream used is 12 per cent.; while if the cream is 16 per cent. the milk required would be 10.8 ounces and the cream 4.8 ounces, and yet the actual percentages of the fat and proteids in the two mixtures would be the same.

The member of the household to whom the modification of the milk is intrusted, and to whom the technique of the modification is explained, should first be warned that she should always endeavor to prevent impurities from getting into the milk, in preference to trying to eradicate them after they have begun to alter the normal composition of the milk. She should conscientiously carry out to the minutest detail the directions which are given to her. The milk of a herd of cows is preferable to that of one cow, for many of the reasons already spoken of, but especially because the elemental percentages are less likely to vary in the mixed milk of a herd than in that of the individual cow, and because the mixing lessens the deleterious effects on the milk arising from occasional disturbances of health in an individual member of the herd. The cows, if possible, should be of a common breed and such as give a moderately rich milk.

The milk should be received into absolutely clean receptacles, thoroughly strained, and rapidly transferred to a clean and cold place free from dust.

It has been shown by Freeman that when milk is set the great mass of the bacteria rise with the cream, so that the separated milk is comparatively free from bacteria, and thus becomes bacteriologically the best material for obtaining the required percentage of proteids in a prescribed mixture; the necessity for pasteurization is thus also greatly diminished, so far as the milk is concerned.

MATERIALS FOR HOME MODIFICATION.—Certain materials and apparatus are required to modify a milk in the home. To obtain the required percentages of the fat resource is chiefly had to creams of various strengths; the sugar is supplied by commercial milk-sugar; the proteids are derived either from whole milk or separated milk, the caseinogen percentage being obtained from milk and the lactalbumin mostly from whey. The alkalinity can best be regulated by freshly prepared lime-water. The water should be freshly distilled or clear water filtered and boiled.

DEFINITION OF TERMS.—For the purpose of uniformity it is well that the meanings of the terms used in speaking of the materials required for a modification of milk should be clearly defined.

Separated milk means a milk from which the fat has been partially or wholly removed, either by the centrifuge or by gravity.

Fat-free milk means a separated milk which contains no fat, or at least only a fractional percentage.

Cream means that which remains after separated milk has been removed from whole milk. It contains most of the fat of the whole milk and certain percentages of all the other elements of whole milk. Its proteid percentage is only slightly lower than that of whole milk, the higher fat percentage creams showing the greater variations, and none of the creams in ordinary use showing a proteid percentage as great as 1 per cent. below that of the whole milk from which they are derived. The sugar percentage is slightly lower than in milk, and the salts are slightly diminished.

Whole milk, therefore, can be considered a 4 per cent. cream.

Whey means a milk which contains all its sugar and water while most of the fat and all the caseinogen have been removed. The fat percentage is therefore very low, and the proteid percentage as a whole is low, the remaining proteids consisting almost entirely of lactalbumin.

Caseinogen is the mother substance from which the casein is obtained by precipitation or coagulation.

Lactalbumin is the proteid not coagulable by acids and rennet.

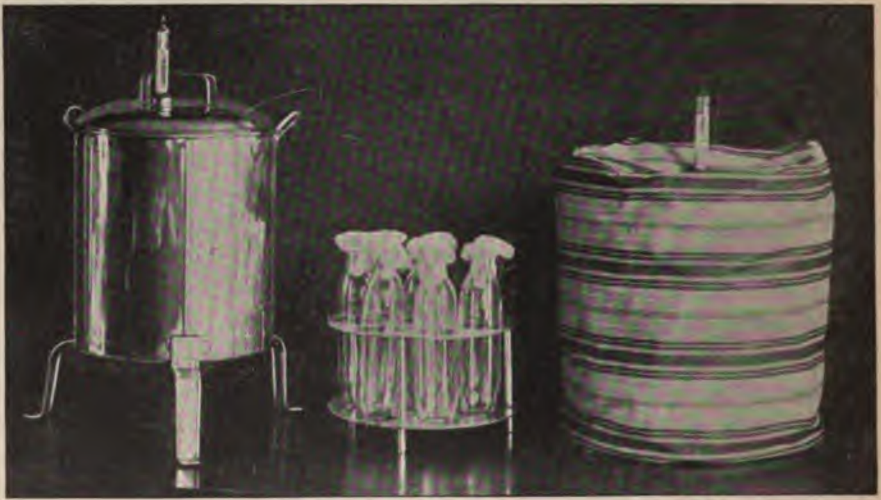
Average Analysis of Whole Milk, 16 per cent. Cream, and Whey.

	Whole Milk.	16 per cent. Cream.	Whey.
	Per Cent.	Per Cent.	Per Cent.
Fat	4.00	16.00	0.32
Sugar	4.50	4.00	4.79
Proteids	4.00	3.60	0.86

APPARATUS.—The apparatus used in the home modification of milk is as follows:

Home-Sterilizer.—If great precautions are taken to guard the milk and cream, they (especially the separated milk) need not be pasteurized or sterilized. In many cases, however, the physician may deem it safer to heat the milk, especially when he is treating a case of gastro-enteric disturbance, or when in hot weather it is necessary to transport the food a long distance. If heating is ordered, a special apparatus will be required This apparatus, which is called a home-sterilizer, can be obtained at the laboratories, or it can be readily devised in the home from a tin pail. Fig. 55 represents the home-sterilizer. It is simply a tin can supported on its legs so that it can be heated by a lamp, or, if preferable, the legs can be removed and the can placed on a stove.

FIG. 55.



Sterilizer and thermometer. Stand for tubes. Sterilizer covered with cozy after removal from heat.

Thermometer.—The sterilizer has a lid, to which is fitted a thermometer by which the degree of heat within the can is indicated.

Tubes.—The tubes, varying in number according to the number of feedings which are required in twenty-four hours, are placed in this stand, which can be lowered into the sterilizer until the water therein is made to rise as high as the level of the milk in the tubes.

Stoppers.—The tubes are stopped with cotton-wool.

Cozy.—The sterilizer is covered with a thick cozy, through which the thermometer from the lid passes and indicates the degree of heat retained within the sterilizer after the flame has been removed.

Graduate.—A 250 c.c. (8½ ounces) glass graduate, divided into half-drachms, will be needed.

Cotton-Wool.—A roll of aseptic non-absorbent cotton-wool should be provided.

Milk-Sugar.—Milk-sugar must be used.

Sugar-Measure.—A sugar-measure, which holds 13.5 grammes (3⅜ drachms), can be obtained at the laboratories.

Fig. 56.

Sugar-measure.

This measure obviates the expense of having the milk-sugar put up in packages by the apothecary, and is sufficiently exact to regulate the sugar percentage in the mixtures which will presently be referred to. It is well to remember, however, that a pound of milk-sugar contains 464 grammes (7000 grains), and that if it is preferred to order the sugar in packages of 13.5 grammes (3⅜ drachms) directly from the apothecary, in place of using the measure, one can simply tell him to make thirty-five packages from the pound, and the mother can then be directed to use a package of milk-sugar instead of a measureful.

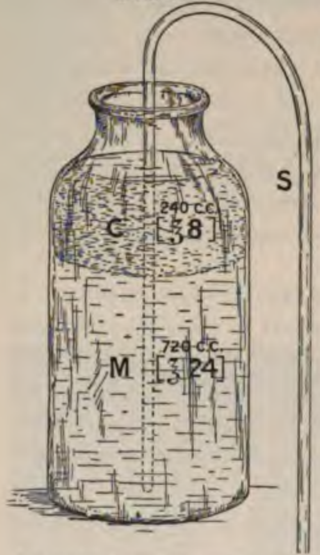
Siphon.—Finally, a glass siphon, 0.6 cm. (¼ inch) caliber, is needed. The siphon can be used in any quart glass jars which the family happen to have.

The siphon should be a glass tube one-quarter to one-half inch in diameter. It can be bent in a gas-flame. The end out of which the milk is to flow should be at least six inches longer than that which is to be inserted in the jar. To operate the siphon, fill it with boiled water, close the longer end with the finger, invert the siphon, and place the shorter end in the milk. Then withdraw the finger, and the water, followed by the milk, will run out of the long arm of the siphon. The mouth should never be used to start the siphon under any circumstances, as pathogenic organisms might in this way be introduced into the milk.

Method of Obtaining Cream and Separated Milk.—In order to obtain the separated milk and the creams of different percentages the jars of milk are to be set in a vessel containing ice and water with some salt in the proportion of 5 grammes (1 teaspoonful) to 960 c.c. (1 quart) of water. Clean, freshly-boiled cotton cloths are then thrown over the uncovered jars. The mouths of the jars are kept open for about fifteen minutes to dispose of the animal heat. The jars are then to be sealed tightly, as is done in preserving, and are left in the ice-water for a variable number of hours according to the fat percentage of cream desired. Care must be taken that the temperature of the water does not fall below 1.66° C. (35° F.).

Fig. 57 represents one of the jars which has been set for six hours with the siphon in position for siphoning out the lower 240 c.c. (8 ounces) of fat-free milk. The upper 240 c.c. (8 ounces) in the jar represent a cream of 10 per cent., and the intermediate 480 c.c. (16 ounces) contain a certain percentage (about 2 per cent.) of fat which in six hours has not separated from the milk.

Fig. 57.



Jar containing milk, cream, and siphon. C, cream; M, milk; S, siphon.

The fat percentage of the cream and the number of ounces of cream to be obtained from the top of the jar depends upon the number of hours of the setting, under the surrounding conditions, and also upon the percentage of fat in the milk which is set.

Prescriptions for Home Modification.—With the required cream siphoned from the top of the jar and the fat-free separated milk siphoned from the bottom can now be obtained the combination of approximate percentages prescribed for the especial case.

These combinations must all be deduced from mathematical formulæ, and the physician can either do this himself in each case or can make use of the following tables, which I have had prepared for this purpose.

The prescriptions have been calculated from a fat-free milk and a cream of 10 per cent. fat, obtained as shown in Fig. 57. The calculations are made for a mixture of 20 ounces. The required percentages of the fat, sugar, and proteids have first been tabulated and then the corresponding amounts of milk, cream, lime-water, water, and sugar placed opposite them. In all the following prescriptions the milk-sugar is to be thoroughly dissolved in the water of the mixture before the other materials are added.

Prescription 28.

Fat	0.25	Cream, 10 per cent	½ ounce.
Sugar	4.00	Fat-free milk	1 ounce.
Proteids	0.25	Lime-water	1 ounce.
Lime-water	5.00	Water	17½ ounces.
			20 ounces.
		Milk-sugar	2 measures.

Prescription 29.

Fat	0.50	Cream, 10 per cent	1 ounce.
Sugar	4.50	Fat-free milk	¾ ounce.
Proteids	0.50	Lime-water	1 ounce.
Lime-water	5.00	Water	17¼ ounces.
			20 ounces.
		Milk-sugar	1¾ measures.

PRESCRIPTION 30.

Fat	1.00	Cream	2 ounces.
Sugar	5.00	Fat-free milk	2 ounces.
Proteids	0.75	Lime-water	1 ounce.
Lime-water	5.00	Water	15 ounces.
			20 ounces.
		Milk-Sugar	2 measures.

PRESCRIPTION 31.

Fat	2.00	Cream	4 ounces.
Sugar	5.00	Fat-free milk	None.
Proteids	0.75	Lime-water	1 ounce
Lime-water	5.00	Water	15 ounces.
			20 ounces.
		Milk-sugar	2 measures.

PRESCRIPTION 32.

Fat	2.00	Cream	4 ounces.
Sugar	5.50	Fat-free milk	1½ ounces.
Proteids	1.00	Lime-water	1 ounce.
Lime-water	5.00	Water	13½ ounces.
			20 ounces.
		Milk-sugar	2¼ measures.

PRESCRIPTION 33.

Fat	2.50	Cream	5 ounces.
Sugar	6.00	Fat-free milk	None.
Proteids	1.00	Lime-water	1 ounce.
Lime-water	5.00	Water	14 ounces.
			20 ounces.
		Milk-sugar	2½ measures.

PRESCRIPTION 34.

Fat	3.50	Cream	7 ounces.
Sugar	6.50	Fat-free milk	1 ounce.
Proteids	1.50	Lime-water	1 ounce.
Lime-water	5.00	Water	11 ounces.
			20 ounces.
		Milk-sugar	2½ measures.

PRESCRIPTION 35.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Fat-free milk	None.
Proteids	1.50	Lime-water	1 ounce.
Lime-water	5.00	Water	11 ounces.
			20 ounces.
		Milk-sugar	2¾ measures.

PRESCRIPTION 36.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Fat-free milk	2½ ounces.
Proteids	2.00	Lime-water	1 ounce.
Lime-water	5.00	Water	8½ ounces.
			20 ounces.
		Milk-sugar	2½ measures.

PRESCRIPTION 37.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Fat-free milk	5 ounces.
Proteids	2.50	Lime-water	1 ounce.
Lime-water	5.00	Water	6 ounces.
			20 ounces.
		Milk-sugar	2¼ measures.

PRESCRIPTION 38.

Fat	4.00	Cream	8 ounces.
Sugar	7.00	Fat-free milk	7½ ounces.
Proteids	3.00	Lime-water	1 ounce.
Lime-water	5.00	Water	3½ ounces.
			20 ounces.
		Milk-sugar	2 measures

PRESCRIPTION 39.

For weaning.

Fat	4.00	Cream	8 ounces.
Sugar	5.00	Fat-free milk	7½ ounces.
Proteids	3.00	Lime-water	1 ounce.
Lime-water	5.00	Water	3½ ounces.
			20 ounces.
		Milk-sugar	1 measure.

PRESCRIPTION 40.

For weaning.

Fat	4.00	Cream	8 ounces.
Sugar	5.00	Fat-free milk	8 ounces.
Proteids	3.25	Lime-Water	1 ounce.
Lime-water	5.00	Water	3 ounces.
			20 ounces.
		Milk-sugar	⅞ measure.

PRESCRIPTION 41.

For weaning.

Fat	4.00	Cream	8 ounces.
Sugar	4.50	Fat-free milk	12 ounces.
Proteids	3.50		20 ounces.

After the various materials have been mixed, in the proportions shown in the formulæ, the requisite amount of food for one feeding is poured into each of the tubes. If it is desired to pasteurize at 75° C. (167° F.), the tubes are stoppled with cotton-wool, care being taken to have a reasonably tight stopple in and a dry neck to the tubes. The tubes are then placed in the rack and lowered into the sterilizer, and the water in the sterilizer is adjusted to the level of the milk in the tubes. Heat, by means of a lamp or stove, is then applied to the sterilizer, which is watched, with the cover off, until the thermometer shows that the water-bath has reached a point of 77.2° C. (171° F.). The lamp is removed as soon as this temperature is reached, the cover put in place, and the cozy over it. The thermometer should mark a temperature of between 75° C. (167° F.) and 77.6° C. (170° F.) for thirty minutes, at the expiration of which time the tubes are to be removed from the sterilizer, and are to be kept in a cool place, preferably the ice-chest, until needed. A temperature of 75° C. (167° F.) is sufficiently high to coagulate the lactalbumin. The percentage of lactalbumin in cow's milk, however, is so small that there is no macroscopic change in the appearance of the milk when heated to this point. On the other hand, a temperature of 68.3° C. (155° F.) will kill most of the organisms in milk, and it is better to heat to this point so as to avoid coagulating the lactalbumin. When the milk is to be transported long distances, as in ocean voyages, it may be considered best to destroy the undeveloped spores of the organisms. To accomplish this the milk should be boiled three times for ten minutes at intervals of twelve hours.

From what has already been said, it will be readily understood that these formulæ, calculated with a 10 per cent. cream, are entirely insufficient in their range for the treatment of a large number of cases which require a correspondingly large number of combinations of percentages before they can be made to digest and to thrive. It therefore becomes necessary for the physician to be prepared to write his prescriptions for a home modification with a number of different creams, as 10 per cent., 12 per cent., 16 per cent., 20 per cent., and many others; that he should also know how to combine these creams with whole milk, with fat-free milk, with whey, and with cereals. In order to do this he must either, as by using the preceding tables, keep beside him for use the number of ounces required for certain percentage combinations, or he must bear in mind the complete analyses of all the materials used, as well as some mathematical equations by which he can calculate any number of percentage combinations.

Table for the Calculation of a Home Modification.—In order to simplify, so far as possible, the calculation of various cream mixtures for home modification, the following table has been arranged by Dr. Maynard Ladd to show certain commonly used percentage combinations with creams of different strength. The figures are given for twenty-ounce mixtures; if the total quantity used in twenty-four hours is thirty ounces, the quantity of each ingredient should be increased one-half; if forty ounces are required, the quantity of each ingredient should be doubled. It will be noted that the amount of fat-free milk used—that is, the lowest quarter of the milk from which the cream has been raised—varies according to the strength of the cream; whereas the other ingredients are not similarly dependent. The table also shows certain percentage combinations which cannot be made either by a ten per cent. or a twelve per cent. cream.

TABLE 51.

Prescriptions calling for a mixture of twenty ounces.				Cream in ounces.				Fat-free Milk in ounces. used with Creams of				Lime-water in ounces.	Boiled water in ounces.	Milk-sugar in measures.
Fats.	Sugar.	Proteids.	Lime-water. Per cent.	Ten per cent.	Twelve per cent.	Sixteen per cent.	Twenty per cent.	Ten per cent.	Twelve per cent.	Sixteen per cent.	Twenty per cent.			
0.50	5.00	2.00	5	1	¾	¾	½	9¼	9½	9½	9¾	1	8¾	1⅓
0.75	6.00	1.00	5	1½	1¼	1	¾	3½	3¾	4	4¼	1	14	2¼
1.00	5.00	0.75	5	2	1½	1¼	1	2	2½	2½	3	1	15	2
1.50	4.00	0.50	5	*	2½	2	1½	*	¼	¾	1¼	1	16¾	1½
2.00	5.00	0.75	5	4	3	2½	1¾	0	1	1½	2¼	1	15	2
2.00	5.50	1.00	5	4	3	2½	1¾	1½	2¼	2¾	3½	1	13½	2¼
2.50	6.00	1.00	5	5	4	3¼	2½	0	1½	2¼	3	1	14	2½
3.00	6.00	0.50	5	*	*	3¾	3	*	*	0	¾	1	15¼	1¾
3.00	6.00	0.75	5	*	5	3¾	3	*	0	1¼	2	1	14	2½
3.00	6.00	1.00	5	*	4¾	3¾	2¾	*	¾	1¾	2¾	1	13½	2¼
3.50	6.50	1.00	5	*	5½	4½	3½	*	0	1	2	1	13½	2½
3.50	6.50	1.50	5	7	5½	4½	3½	1	2½	3½	4½	1	11	2½
3.00	7.00	1.00	5	*	4¾	3¾	2¾	*	¾	1¾	2¾	1	13½	2¾
3.00	7.00	1.50	5	6	4¾	3¾	2¾	2	3¼	4¼	5¼	1	11	2½
3.00	7.00	2.00	5	6	4¾	3¾	2¾	4½	5¾	6¾	7¾	1	8¾	2½
4.00	7.00	1.00	5	*	*	5	3¾	*	*	½	1¾	1	13½	2¾
4.00	7.00	1.50	5	8	6¼	5	3¾	0	1¾	3	4¼	1	11	2¾
4.00	7.00	2.00	5	8	6¼	5	3¾	2½	4¼	5½	6¾	1	8½	2½
4.00	7.00	2.50	5	8	6¼	5	3¾	5	6¾	8	9¼	1	6	2
4.00	7.00	3.00	5	8	6¼	5	3¾	7½	9¼	10½	11¾	1	3½	2
4.00	6.00	3.00	5	8	6¼	5	3¾	7½	9¼	10½	11¾	1	3½	1¼
4.00	5.00	3.00	5	8	6¼	5	3¾	7½	9¼	10½	11¾	1	3½	1
4.00	5.00	3.50	5	8	6¼	5	3¾	10	11¾	13	14¼	1	1	⅓

* Impossible combination with the percentage of cream indicated.

In using this table, accurate results can be obtained if the percentage of cream is known and a guaranteed cream can be purchased from the laboratory. If this is impracticable, the physician must use his ingenuity in estimating the fat percentage of cream, and such estimation can, of course, only be approximate, as is clearly shown by the widely divergent results obtained from setting different specimens of milk, as described on page 221. However, as a basis on which to form an *approximate* estimation we may assume that a milk of average strength—that is, one with fat 4 per cent., sugar 4.50 per cent., proteid 4 per cent.,—will yield, if it has not been previously setting, and if placed in a temperature of about 3.3° C. (38° F.), a top quarter (eight ounces in each quart of milk) of cream containing 10 per cent. fat at the end of six hours, or 12 per cent. fat at the end of eight hours. A gravity cream containing 16 per cent. fat can be obtained under very favorable conditions by setting the milk twelve hours, but it is safer not to count on so high a percentage. Milk which has been setting eight hours will yield a more uniform percentage of cream than one which has been setting six hours. If the milk which is used is known to be exceptionally low in fat, as in certain herds, it would be safer to let it set eight hours and calculate on a 10 per cent. instead of a 12 per cent. cream in the uppermost eight ounces. If the milk is known to be exceptionally rich in fat, it is more likely at the end of six hours to yield a 12 per cent. cream than a 10 per cent. cream. Each measure of sugar may be approximately estimated to be equivalent to a level tablespoonful. It is better, however, to buy a measure such as is described on page 225.

THE THEORY OF PERCENTAGE MODIFICATION.—Dilution of Creams with Whole Milk.—Although a number of combinations of various percentages of fat and sugar can be obtained by diluting creams with whole milk, yet, the proteid percentage in any instance does not bear the same ratio to the fat percentage as holds in the cream from which the dilution is made. The finer variations in the relative proportions of fat and proteids which are easily obtained by systematized modification in the laboratories are impossible by the simple dilution of cream.

The following table shows the fat percentages of cream which can be obtained by diluting a 20 per cent. cream with whole milk.

TABLE 52.

1 part 20 per cent. cream + 3 parts whole milk = 8 per cent. cream.
1 part 20 per cent. cream + 1 part whole milk = 12 per cent. cream.
3 parts 20 per cent. cream + 1 part whole milk = 16 per cent. cream.

Dilutions of Cream with Water.—If cream of definite percentages of fat, sugar, and proteids are diluted with equal or multiple quantities of water, various mixtures are obtained in which the percentages of these three elements bear a fixed ratio to those of the cream used. (Westcott.) The percentages of the dilution can easily be calculated by multiplying the percentages of the cream by a fraction of which the numerator is the integer representing the quantity of cream, and the denominator the integer representing the sum of the quantity of the cream and of the diluent. Thus, for an 8 per cent. cream with percentages of 8 per cent. fat, 4.40 per cent. sugar, and 3.90 per cent. proteids, a mixture of equal parts cream and water would give 4 per cent. fat and 1.95 per cent. proteids. The resulting sugar percentage would also bear the same fixed ratio to that of the sugar percentage in the cream.

Dilutions of Cream with Solutions of Sugar.—If it is desired to obtain various combinations of fat, sugar, and proteids by diluting creams with solutions of sugar, the various percentages of sugar solution can be obtained by dissolving one ounce of milk-sugar in twenty ounces, sixteen and one-half ounces, fourteen and one-quarter ounces, twelve and one-half ounces, or ten ounces of boiled or distilled water, which results respectively in sugar solutions of 5, 6, 7, 8, or 10 per cent. (Westcott.) When these sugar solutions are used as the diluent the calculation of the resulting sugar percentage can be made by the following formula, which expresses the fact that the sugar percentage of the dilution is the sum of the percentages contributed by the sugar solution and the cream.

$$S = \frac{W \times s' + (C \times c)}{W + C}.$$

In this equation S represents the resultant sugar percentage; W represents the quantity of water; s′ represents the percentage of the sugar solution;

c represents the percentage of sugar in the cream; and C represents the quantity of cream.

For example, for a dilution of 1 part of 12 per cent. cream to 3 parts of an 8 per cent. sugar solution the resulting percentages would be 3 per cent. fat and 0.95 per cent. proteids, while the sugar percentage would be

$$S = \frac{3 \times 8 + (1 \times 4.20)}{3 + 1} = \frac{28.20}{4} = 7.05.$$

The following tables have been prepared by Dr. T. S. Westcott to show the different resulting fat, sugar, and proteid percentages obtained by mixing in various proportions different sugar percentage solutions with 4 per cent. fat cream (*i.e.*, whole milk) 8 per cent. fat cream, 12 per cent. fat cream, and 16 per cent. fat cream.

TABLE 53.

3 parts of milk to
1 part 5 to 10 per cent. sugar solution = fat, 3.00; sugar, 4.60 to 5.85; proteids, 3.00

TABLE 54.

Four per cent. Cream (whole milk).

Fat, 4.00; Sugar, 4.40; Proteids, 4.00.

1 part of milk to
11 parts 5 to 7 per cent. sugar solution = fat, 0.33; sugar, 4.95 to 6.78; proteids, 0.33
7 parts 5 to 7 per cent. sugar solution = fat, 0.50; sugar, 4.92 to 6.67; proteids, 0.50
3 parts 5 to 8 per cent. sugar solution = fat, 1.00; sugar, 4.85 to 7.10; proteids, 1.00
1 part 5 to 10 per cent. sugar solution = fat, 2.00; sugar, 4.70 to 7.20; proteids, 2.00

TABLE 55.

Eight per cent. Cream.

Fat, 8.00; Sugar, 4.30; Proteids, 3.90.

1 part of cream to
7 parts 5 to 7 per cent. sugar solution = fat, 1.00; sugar, 4.91 to 6.66; proteids, 0.49
3 parts 5 to 8 per cent. sugar solution = fat, 2.00; sugar, 4.82 to 7.07; proteids, 0.97
16 parts 5 to 8 per cent. sugar solution = fat, 3.07; sugar, 4.73 to 6.58; proteids, 1.41
1 part 5 to 10 per cent. sugar solution = fat, 4.00; sugar, 4.65 to 7.15; proteids, 1.95

TABLE 56.

Twelve per cent. Cream.

Fat, 12.00; Sugar, 4.20; Proteids, 3.80.

1 part of cream to
11 parts 5 per cent. sugar solution = fat, 1.00; sugar, 4.93; proteids, 0.32
11 parts 6 per cent. sugar solution = fat, 1.00; sugar, 5.85; proteids, 0.32
11 parts 7 per cent. sugar solution = fat, 1.00; sugar, 6.76; proteids, 0.32
7 parts 5 to 7 per cent. sugar solution = fat, 1.50; sugar, 4.90 to 6.65; proteids, 0.48
5 parts 5 to 7 per cent. sugar solution = fat, 2.00; sugar, 4.87 to 6.53; proteids, 0.63
3.8 parts 5 to 8 per cent. sugar solution = fat, 2.50; sugar, 4.83 to 7.20; proteids, 0.79
3 parts 5 to 8 per cent. sugar solution = fat, 3.00; sugar, 4.80 to 7.05; proteids, 0.95
2.4 parts 5 to 8 per cent. sugar solution = fat, 3.53; sugar, 4.76 to 6.88; proteids, 1.12
2 parts 5 to 8 per cent. sugar solution = fat, 4.00; sugar, 4.73 to 6.73; proteids, 1.27

TABLE 57.

Sixteen per cent. Cream.

Fat, 16.00; Sugar, 4.00; Proteids, 3.60.

1 part of cream to

15 parts 5 per cent. sugar solution = fat, 1.00; sugar, 4.94; proteids, 0.23
15 parts 6 per cent. sugar solution = fat, 1.00; sugar, 5.87; proteids, 0.23
15 parts 7 per cent. sugar solution = fat, 1.00; sugar, 6.81; proteids, 0.23
9 parts 5 per cent. sugar solution = fat, 1.60; sugar, 4.90; proteids, 0.36
9 parts 6 per cent. sugar solution = fat, 1.60; sugar, 5.80; proteids, 0.36
9 parts 7 per cent. sugar solution = fat, 1.60; sugar, 6.70; proteids, 0.36
7 parts 5 per cent. sugar solution = fat, 2.00; sugar, 4.87; proteids, 0.45
7 parts 6 per cent. sugar solution = fat, 2.00; sugar, 5.75; proteids, 0.45
7 parts 7 per cent. sugar solution = fat, 2.00; sugar, 6.62; proteids, 0.45
5.4 parts 5 per cent. sugar solution = fat, 2.50; sugar, 4.84; proteids, 0.56
5.4 parts 6 per cent. sugar solution = fat, 2.50; sugar, 5.70; proteids, 0.56
5.4 parts 7 per cent. sugar solution = fat, 2.50; sugar, 6.53; proteids, 0.56
4.3 parts 5 per cent. sugar solution = fat, 3.02; sugar, 4.81; proteids, 0.68
4.3 parts 6 per cent. sugar solution = fat, 3.02; sugar, 5.62; proteids, 0.68
4.3 parts 7 per cent. sugar solution = fat, 3.02; sugar, 6.43; proteids, 0.68
3.6 parts 5 per cent. sugar solution = fat, 3.48; sugar, 4.78; proteids, 0.78
3.6 parts 6 per cent. sugar solution = fat, 3.48; sugar, 5.56; proteids, 0.78
3.6 parts 7 per cent. sugar solution = fat, 3.48; sugar, 6.35; proteids, 0.78
3 parts 5 per cent. sugar solution = fat, 4.00; sugar, 4.75; proteids, 0.90
3 parts 6 per cent. sugar solution = fat, 4.00; sugar, 5.50; proteids, 0.90
3 parts 7 per cent. sugar solution = fat, 4.00; sugar, 6.25; proteids, 0.90
3 parts 8 per cent. sugar solution = fat, 4.00; sugar, 7.00; proteids, 0.90

DILUTIONS OF CREAMS WITH WHEY.—When it is desired to increase in a mixture the relative proportion of the lactalbumin to the caseinogen, whey can be used for this purpose. There are, however, in doing this, certain precautions which must be taken, since the excess of pepsin resulting from the preparation of the whey is apt to precipitate the caseinogen of the proteids in the cream, and to leave coagula in the mixture. To obviate this the whey should be raised to a temperature of 65.5° C. (150° F.) for five minutes in order to destroy the rennin, and then allowed to cool before mixing it with the cream, especially when the total quantity of food for twenty-four hours is prepared at one time. This procedure, however, seems to be unnecessary when the whey is added to cream and milk mixtures which have been partially peptonized. In this case the amount of whey to be added should first be decided upon and then this quantity should be deducted from the quantity of diluent used, so that when the whey is added the total quantity shall not be altered. If this be done, the increase in proteids (0.86) contributed by the whey can be calculated by multiplying 0.86 by the number of ounces of whey and dividing the product by the total number of ounces in the prescribed mixture. Since the proteid (lactalbumin) percentage of whey is less than 1 per cent., a very nutritious mixture may be obtained by using undiluted whey and bringing up the fat percentage by the addition of cream.

In this case, since there is no diluent, the fat percentage cannot be varied altogether at will, but must depend upon the desired proteid percentage and *vice versa*.

The proteid percentage, however, for any definite fat percentage can be varied by making use of different grades of cream. Thus, with a 3 per cent. fat (F), proteids ranging from 1.18 (from a 20 per cent. cream) up to 1.92 (from an 8 per cent. cream) can be obtained, and with a 4 per cent. fat (F) a variation of proteids between 1.30 per cent. and 2.32 per cent. can be similarly obtained.

The formula by which the amount of cream (C) to be used in a total mixture (Q) in which the diluent is whey is as follows:

$$C = \frac{Q[F(\text{fat p. c. in cream used}) - 0.32\ (\text{fat p. c. in whey})]}{7.68 \text{ or } 11.68 \text{ or } 15.68 \text{ or } 19.68}$$

according as a cream of fat 8 per cent., 12 per cent., 16 per cent., or 20 per cent. is used.

The following table gives the varying combinations of fat and proteids that can be obtained by mixtures of whey and creams of various fat percentages. For the sake of comparison the quantities of cream required for a mixture of twenty ounces have been given in the final column:

TABLE 58.

Combinations of fat and proteid percentages which can be made by creams of 4 per cent., 8 per cent., 12 per cent., 16 per cent., and 20 per cent. diluted with whey, containing a fat percentage of 0.32.

		CREAM IN TWENTY-OUNCE MIXTURE, THE BALANCE WHEY.
WITH 20 PER CENT. CREAM.		
For 1.00 F.	P. = 0.94	0.70 ounce.
For 2.00 F.	P. = 1.06	1.71 ounces.
For 3.00 F.	P. = 1.18	2.72 ounces.
For 4.00 F.	P. = 1.30	3.74 ounces.
WITH 16 PER CENT. CREAM.		
For 1.00 F.	P. = 0.98	0.87 ounce.
For 2.00 F.	P. = 1.15	2.14 ounces.
For 3.00 F.	P. = 1.32	3.42 ounces.
For 4.00 F.	P. = 1.50	4.69 ounces.
WITH 12 PER CENT. CREAM.		
For 1.00 F.	P. = 1.03	1.16 ounces.
For 2.00 F.	P. = 1.28	2.88 ounces.
For 3.00 F.	P. = 1.53	4.59 ounces.
For 4.00 F.	P. = 1.79	6.30 ounces.
WITH 8 PER CENT. CREAM.		
For 1.00 F.	P. = 1.18	1.77 ounces.
For 2.00 F.	P. = 1.53	4.38 ounces.
For 3.00 F.	P. = 1.92	6.98 ounces.
For 4.00 F.	P. = 2.32	9.58 ounces.
WITH 4 PER CENT. CREAM (MILK).		
For 1.00 F.	P. = 1.44	3.69 ounces.
For 2.00 F.	P. = 2.29	9.13 ounces.
For 3.00 F.	P. = 3.15	14.56 ounces.
For 4.00 F.	P. = 4.00	20.00 ounces.

Preparation of Sweet Whey.—Sweet whey is best made by the following method: For each pint of whey needed, take one quart of whole fresh milk or fat-free milk, heated to 37.7° C. (100° F.), and add 8 c.c. (2 drachms) of the essence of pepsin or some of the preparations of liquid rennet. This will precipitate the casein in the form of a curd, which is then broken up with a fork, and the fluid which remains is the whey. This is strained through two thicknesses of boiled cheese-cloth and one thickness of absorbent cotton and slowly cooled to a temperature of 10° C. (50° F.), and kept on ice until needed. If the whey is to be mixed with cream, it must first be heated to 65.5° C. (150° F.), in order to kill the rennet enzyme. Whey mixtures should not be heated above 68.3° C. (155° F.) if one wishes to keep safely under the coagulation point of the lactalbumin.

General Formulæ for Calculation of all Percentage Combinations.—The following formulæ and equations, calculated by Westcott, can be used for obtaining any combinations of the percentages of the fat, sugar, and proteids of milk prescribed, provided that creams of varied fat percentages are used, as has been already explained. The mixtures with cream may be made with whole milk, with fat-free milk, and with whey. The following symbols will be used:

F = prescribed percentage of fat.
S = prescribed percentage of sugar.
P = prescribed percentage of proteids.
C = total quantity of cream in ounces.
M = total quantity of milk in ounces.
W = total quantity of water in ounces.
L = total quantity of dry milk-sugar in ounces.
Q = total quantity of mixture.
a and a' = known percentage of fat in cream and milk respectively.
b and b' = known percentage of proteids in cream and milk respectively.
c and c' = known percentage of sugar in cream and milk respectively.

Since the actual quantity of proteids in a percentage mixture is the sum of the quantities of proteids contributed by the milk and the cream, and, again, since the actual quantity of fat is the sum of the quantities of fat contributed by the milk and the cream, the following fundamental formulæ represent these facts. Since P, F, etc., are represented by integers instead of hundredths, the following equations (1) and (2) should for greater accuracy be expressed as divided by 100, as already expressed in the formula for sugar on page 237; but since both sides of the equation are divided by 100, the equality of the numerators is also true.

$$Q \times P = b' M + b C. \tag{1}$$

$$Q \times F = a' M + a C. \tag{2}$$

And since the same reasoning applies to the actual quantity of sugar in the mixture with the addition of the dry sugar needed to bring the percentage up to the percentage of sugar prescribed, we have

$$Q \times \frac{S}{100} = \frac{c' M}{100} + \frac{c C}{100} + L. \tag{3}$$

Transposing (1), so as to get a value for M, we obtain

$$M = \frac{Q P - b C}{b'}. \tag{4}$$

If this value of M is substituted in equation (2) we have, by transposition and collecting,

$$C = \frac{Q (b' F - a' P)}{a b' - a' b}. \tag{5}$$

By finding a value for M, from (2) instead of (1) we have

$$M = \frac{Q F - a C}{a'}. \tag{6}$$

From equation (3), by transposition, we obtain

$$L = \frac{Q S - (c' M + c C)}{100}. \tag{7}$$

These formulæ are of universal application for any strengths of the cream and milk percentages.

Formulæ for Cream and Whole Milk (4 per cent. Cream).—Thus, for a combination of 16 per cent. cream ($a = 16$, $b = 3.6$, $S = 4$), and 4 per cent. milk ($a' = 4$, $b' = 4$, $S = 4.40$), as a′ and b′ are equal, this value can be taken out of the numerator and the denominator of (5), leaving

$$C = \frac{Q (F - P)}{a - b}. \tag{8}$$

Substituting values for a and b, the formula becomes

$$C = \frac{Q (F - P)}{16 - 3.6} = \frac{Q (F - P)}{12.4} \tag{9}$$

and formula (6) becomes

$$M = \frac{Q F - 16 C}{4} = \frac{Q F}{4} - 4 C. \tag{10}$$

In the same way, for a 12 per cent. cream ($a = 12, b = 3.8, S = 4.20$) and a 4 per cent. milk, formula (8) becomes

$$C = \frac{Q (F - P)}{12 - 3.8} = \frac{Q (F - P)}{8.2} \tag{11}$$

and (6) becomes

$$M = \frac{Q\,F - 12\,C}{4} = \frac{Q\,F}{4} - 3\,C. \tag{12}$$

Similarly, for 10 per cent. cream (a = 10, b = 3.85, S = 4.25) formula (8) becomes

$$C = \frac{Q\,(F - P)}{10 - 3.85} = \frac{Q\,(F - P)}{6.15}, \tag{13}$$

$$M = \frac{Q\,F}{4} - \frac{10\,C}{4}. \tag{14}$$

Formulæ for Cream and Fat-free Milk.—When in place of mixing the cream with whole milk a fat-free milk is used, we take formula (5) and substituting 0 for the fat value (a′) of the milk we get

$$C = \frac{Q\,(b'\,F - 0 \times P)}{a\,b' - 0 \times b} = \frac{Q\,b'\,F}{a\,b'} = \frac{Q\,F}{a}, \tag{15}$$

and from (4) we get

$$M = \frac{Q\,P - b\,C}{b'}. \tag{16}$$

Thus, for 16 per cent. cream and fat-free milk (15) becomes

$$C = \frac{Q \times F}{16} \text{ or } \frac{F}{16} \times Q, \tag{17}$$

and (16) becomes

$$M = \frac{Q\,P - 3.60\,C}{4}. \tag{18}$$

In the same way, with 12 per cent. cream,

$$C = \frac{F}{12} \times Q. \tag{19}$$

$$M = \frac{Q\,P - 3.80\,C}{4}. \tag{20}$$

In the same way, with 10 per cent. cream,

$$C = \frac{F}{10} \times Q. \tag{21}$$

$$M = \frac{Q\,P - 3.85\,C}{4}. \tag{22}$$

The formula for sugar does not vary much. Formula (7) is of universal application, and, assuming the sugar percentage of milk and cream to be about 4.40, (7) would become

$$L = \frac{Q\,S - 4.40\,(M - C)}{100}. \tag{23}$$

It must be remembered when using these general formulæ for any combination of percentages, as, for instance, in Formula 8, that when P = F it is evident that C = 0; or, in other words, that the mixture becomes a simple dilution of milk, which gives equal percentages of proteids and fat. When P is greater than F, the value of C becomes a negative quantity, which indicates that the milk needs the addition of proteids without fat. This calls for fat-free milk. The calculations of the quantities for such a mixture can be carried out by the formulæ given in the various formulæ derived from (15) and (16).

Formulæ for Cream and Whey.—In order to calculate the amount of whey which is needed for various combinations, the general formulæ (5), (6), and (7) can be applied by considering whey as a milk containing very low proteids (lactalbumin) and fat. Taking König's formula for whey as a standard,

Fat.............................. 0.32 = a′
Sugar............................ 4.79 = c′
Proteids......................... 0.86 = d′

we can then represent a′ of the general formula by 0.32, b′ by 0.86, and c′ by 4.8, and the special formula will then be

$$\text{(24)} \qquad C = \frac{Q\,(0.86 \times F - 0.32 \times P)}{9.1 \text{ or } 12.6},$$

according as twelve per cent. or sixteen per cent. cream is used.

$$\text{(25)} \qquad \text{Whey} = \frac{Q\,F - 12\,C}{0.32} \text{ or } \frac{Q\,F - 16\,C}{0.82}$$

and

$$\text{(26)} \qquad L = \frac{Q\,S - (4.8 \times \text{whey} + 12 \text{ or } 16\,C)}{100}.$$

In such a combination sufficient diluent must be added to make up the total quantity.

The following formulæ are derivable from equations expressing the fact that the proteid or fat percentage of the mixture is equal to the sum of the proteid or fat percentages contributed by the cream and the whey.

$$\text{(27)} \qquad P = \frac{C}{Q} \times b + \frac{\text{whey}}{Q} \times b'$$

$$\text{(28)} \qquad F = \frac{C}{Q} \times a + \frac{\text{whey}}{Q} \times a'$$

whence, by deduction,

$$\text{(29)} \qquad C = \frac{Q\,(F - a')}{a - a'}.$$

One or the other of these formulæ may be used according as a definite fat or proteid percentage is desired. The constants a and a′ represent the fat percentages of the cream and of the whey respectively, and b and b′ represent the corresponding proteid percentages.

Thus, for 20 per cent. cream (F = 20, P = 3.20, S = 3.80) and whey (F = 0.32, P = 0.86, S = 0.48) the formulæ would become

$$\text{(30)} \qquad C = \frac{Q\,(P - 0.86)}{3.20 - 0.86} = \text{(31)} \frac{Q\,(P - 0.86)}{2.34},$$

and

$$C = \frac{Q\,(F - 0.32)}{20 - 0.32} = (33)\ \frac{Q\,(F - 0.32)}{19.68}. \tag{32}$$

The formula for L can be derived from the general formula (7) by substitution, which gives

$$L = \frac{Q\,S - (4.8\ \text{whey} + 3.8\ C)}{100}. \tag{34}$$

In the same way, for 16 per cent. cream the formulæ become, after substitution,

$$C = \frac{Q\,(P - 0.86)}{2.74}, \tag{35}$$

$$C = \frac{Q\,(F - 0.32)}{15.68}. \tag{36}$$

For 12 per cent. cream,

$$C = \frac{Q\,(P - 0.86)}{2.94}, \tag{37}$$

$$C = \frac{Q\,(F - 0.32)}{11.68}. \tag{38}$$

For 8 per cent. cream,

$$C = \frac{Q\,(P - 0.86)}{3.04}, \tag{39}$$

$$C = \frac{Q\,(F - 0.32)}{7.68}. \tag{40}$$

Final Remarks on Home Modification.—If a careful study be made of what has been said under home modification, in comparison with the methods and results of preparing a food at the laboratory, it will be seen how uncertain must be most of the modifications which are made in the homes by mothers and nurses. While, therefore, in many cases the milk must be modified at home, it must be recognized that it probably only approaches in exactness the modification by the clerks at the laboratory, where it is done well with all the factors of the problem taken into consideration, and that in most cases in a home modification the infant is not securing as a food what the physician has prescribed and takes for granted the infant is getting.

Oat-Jelly.—For the preparation of oat-jelly the following method should be employed:

Sixty grammes (2 ounces) of coarse oatmeal are allowed to soak in a quart of cold water for twelve hours. The mixture is then boiled down so as to make a pint, and is strained through a fine cloth while it is hot. When it cools a jelly is formed, which is to be kept on ice until needed. Different proportions of this jelly can be used, but usually it is best to begin with equal parts of jelly and cow's milk. When needed this mixture is warmed and a little salt added.

Barley-Water.—Barley-water is made by boiling 120 grammes (4 ounces) of barley flour in a quart of water for one-half hour, and then straining.

If a barley-jelly is to be made, 120 grammes (4 ounces) of barley flour are employed, and the same process is gone through with as for the preparation of barley-water. The resulting jelly is treated in the same way with milk as I have directed for oat-jelly.

WHEAT.—Wheat can be prepared by the same method as that described for oats and barley.

PEPTONIZED MILK.—Milk may be partially or wholly peptonized in home modification by the same methods as described under laboratory feeding, on page 202.

ARTIFICIAL FOODS FOR INFANTS.—It would seem hardly necessary to suggest that the proper authority for establishing rules for substitute feeding should emanate from the medical profession, and not from non-medical capitalists. Yet, when we study the history of artificial feeding as it is represented all over the world, the position which the family physician occupies, in comparison with that of the venders of the numberless patent and proprietary artificial foods administered by the nurses, is a humiliating one, and should no longer be tolerated.

If we are abreast of the times, if we but recognize and do justice to the work which has lately been done by our own profession, we surely will not hesitate to relegate to oblivion the statements of the food proprietors, which on box and can, on bottle and printed circular, attempt to stem the slow but inevitably progressing wave of scientific investigation.

It may be well to bear in mind that the attempts which in the past have been made to manufacture cheap foods have been marked by failures. We must first, regardless of expense, learn to produce by modification a perfected substitute food, and not endanger the success of our undertaking by allowing the mercantile side of the question to cripple us in the use of costly methods, which, however, we know to be the best. We should, in fact, remember that the human milk, which we are endeavoring to copy, far from being a cheap product, is a very expensive one.

My own opinion in regard to patent foods, as a whole, is that they must necessarily be unreliable. They are thrown on a market where the competition is extreme, and when once they have been advertised into public notice I cannot but feel that irregularities and changes—slight, perhaps, in the eyes of the makers—may unintentionally creep in and carry their composition still farther from that of the standard, human milk.

Analyses show that there is a lack of uniformity in these foods from year to year, and that original claims are apparently forgotten or allowed to give way to cheaper production. In fact, as my experience in the feeding of infants increases, and as I examine year by year the effects of the different foods on infants, I am strongly impressed with the belief that with our present physiological, chemical, and clinical knowledge all the patent foods are entirely unnecessary. The claims made for them are not supported by intelligent and unprejudiced investigation. Those who manufacture them are not in a position to judge correctly concerning them. The merit at times of their apparent success does not belong to them, but to accompanying circumstances. They do great harm by impressing upon the public the false idea that a cheap, easily prepared food is for the good of the infant and is better than anything which can be procured elsewhere. They vary too greatly in their analyses to keep even within the acknowledged varying limits of human milk. It is therefore high time for physicians to appreciate exactly how inefficient in themselves and how misleading in their claims are these artificial foods, and also in what a false position, as the protector of and adviser to the public, our profession is placed whenever it lends itself to even a toleration of their use. I speak of them here simply because there is no doubt that they are kept in the market by the physician rather than by the manufacturer. The latter is only doing what any capitalist interested in a business venture would do. The former, it seems to me, is, perhaps unintentionally, aiding the business interests of others at the expense of his own future reputation as a scientist. It makes little difference to physicians as to what is claimed for these foods when they are placed in the market. It makes a great difference what the mixture contains when given by the mother to the infant according to the directions on the label. For instance, a food may show by its published and certified analysis a fair percentage of fat or sugar, and yet this same food when diluted for the infant's feeding may have these constituents reduced far below the reasonable limits of nutrition.

MATZOON.—Matzoon is a fermented milk made by the action of an imported ferment, probably a form of yeast, upon cow's milk. The milk is first boiled for the purpose of sterilization, the matzoon ferment is added, and the fermentation is begun at a temperature of about 40.5° C. (105° F.) and continued in an open vessel for twelve hours. The temperature is gradually cooled to about 21.1° C. (70° F.) and the milk is bottled and kept on ice. It is ready for use in twenty-four hours. If used for infant feeding, it should be diluted with water and fed with a spoon, as it is too thick to be drawn from a bottle.

KUMYSS.—The best formula for the domestic manufacture of kumyss is that described by Holt: Take one quart of fresh milk, half an ounce of sugar, two ounces of water, and a piece of fresh yeast-cake half an inch square. These materials are put into wired bottles and kept at a temperature of 15.5° C. (60° F.) or 21.1° C. (70° F.) for one week, shaking five or six times a day. The milk is then put upon the ice until used.

MALTED FOODS.—These are obtained by the action of the malt diastase upon wheat and barley flour, and are composed of a mixture of dextrine, dextrose, and maltose, with a small amount of cane-sugar.

Matzoon, kumyss, and malted foods, in the light of recent researches and advances which have been made in the feeding of infants and children with modified milk, may no longer be considered as advisable foods.

SECOND NUTRITIVE PERIOD.

During the eleventh and twelfth months of life the amylolytic function of the infant has become almost fully developed. In accordance with the rule regarding the use of the different functions,—namely, that a function should not be taxed before it is developed, but that when its development is almost completed it should be brought into use,—we should in the latter part of the first year begin to use that function of the digestive tract by means of which the amylaceous elements of the food are converted into sugar.

At this age the percentage of sugar should gradually be reduced in the modified milk and the percentage of proteids increased until the whole milk is found to be digested. The reason for changing the relative percentages of these elements is that the power to digest proteids has much increased during the latter part of the first year. The capacity for digesting a high percentage of sugar is just as great at this period as at an earlier one, but the amount of sugar, given directly as such, in a modified milk, which is required in the later is not so great proportionately as in the earlier period. A large portion of the sugar which is needed for nutrition in this later period is intended to be introduced into the economy by means of a new element in the food,—starch. A certain amount of sugar is, as before, directly introduced into the gastro-enteric tract from the milk-sugar of the milk, and the starch when converted into sugar supplies the remaining portion of sugar needed for nutrition. In a normal infant with normal digestive functions a considerable percentage of starch can be digested and absorbed with benefit in the eleventh and twelfth months. It is well, however, at first not to give immediately a high percentage of starch in the mixture, and also not to ignore the fact that the various cereals contain fat and proteids as well as starch. Consideration must also be paid to what the total resulting percentage of sugar will be when contributed by the converted starch and the milk-sugar in the milk. In this way a gradual change of percentages can be made in the infant's food until its digestive capabilities have become adapted to the food values indicated for digestion in the second year. This indication is for a higher fat, sugar, and proteid percentage in the food and for whole milk, preferably from Holstein cows. When, however, the digestion is weak, a modified milk can be continued into the second year.

Any additional sugar which may be needed with the food can at this time be in the form of cane-sugar rather than milk-sugar. The starch can best be obtained from preparations of oats, barley, and wheat. There is a larger percentage of starch in oats than in barley. It is also more nutritious in every respect, as it contains a considerable percentage of fat. The starch in oats takes a somewhat longer time to be converted into sugar than does that of barley, so that in the case of an infant whose amylolytic function is not fully developed or is somewhat weak, preparations of barley will be better to begin with, because they do not introduce so high a percentage of starch into the food, and also because the starch will be more readily converted into sugar. Preparations of oats seem to be the best form of food to be added to the milk when the infant has reached a period at which it needs a change in the character of its food.

We have, therefore, in preparations of oats, both for purposes of weaning and for establishing a new regimen of diet for the infant, a food which in combination with cow's milk satisfies completely the demands which the digestive functions at this period are making for a perfect nutriment. In using cereals in the food they are best at first reduced to a jelly, as described on page 239.

The second nutritive period may be reckoned to last from the twelfth to the twenty-eighth or thirtieth month of life. That is about the second half of the period which we are in the habit of calling infancy. It also includes the time when the last four teeth of the first set appear. In this second nutritive period the element of variety in the food becomes important. It is undoubtedly important that the actual nutritive values of the food which it is best to give to infants in this period be considered, but it is much more important that special attention be paid to its variety. Foods should be given which while containing a fair percentage of nutritive elements yet differ in the combination of these elements to such a degree that they fulfil the requirements of this period of life. It is best to increase gradually the variety of articles of diet from the twelfth to the twentieth month, always adapting the food to the especial infant. Thus, some infants may be able to digest and assimilate proportionately large quantities of starch; others may both need and digest larger proportions of the proteids or of sugar than the infants first spoken of.

Between the twelfth and thirteenth months the infant should have five meals during the day. At this time it is well to accustom it to take its food from a spoon, and as soon as possible to omit feeding from the bottle. The five meals should be arranged in the following manner:

For breakfast, bread and cow's milk, slightly warmed.

For lunch, equal parts of oat-jelly and cow's milk, warmed, with a little salt added according to the infant's taste.

This meal of oat-jelly should be repeated in the middle of the afternoon.

In the middle of the day, broth of some kind, either chicken or mutton, carefully prepared so as to be free from fat on its surface, can be given with some bread.

The fifth meal should be given in the latter part of the afternoon, and should consist of bread and milk.

In some cases it is impossible to make infants swallow bread for a long period after the usual time of twelve to thirteen months. At times it is not until they are two and one-half to three years old that they can be induced to take bread. In these cases we must feed them according to our judgment of the individual case.

When the infant is fourteen to fifteen months old, some thoroughly boiled rice can be added to the broth in the middle of the day, and if it digests this well it can also have broth given with this meal.

When the infant is sixteen months old, it can have a small amount of butter on its bread. When it is seventeen to eighteen months old, it can have a thoroughly baked white potato, mixed with butter and salt, added to its mid-day meal of bread. When it is nineteen to twenty months old, eggs can become part of its diet.

There are not many fruits which should be given to the infant in its second year. A baked apple can be given at the evening meal when the infant is fourteen to fifteen months old; or, for variety, the apple can be made into a simple sauce, never, however, having the sauce made with much sugar. When peaches are in season, a ripe peach can often be given with benefit, especially if the infant is inclined to be constipated. Other fruits should be avoided, as they are not necessary for the infant's nutrition and at times produce serious trouble.

This is the diet which is sufficient for the infant during the second nutritive period. It is important for the subsequent integrity of the infant's digestion and general nutrition that the parents should insist that no other articles of food be employed, except such as are similar to those which have been mentioned,—namely, the cereals in a variety of forms, according to the taste, judgment, and knowledge of cooking which exists in the special household. For instance, preparations of wheat and barley cooked in various forms may be given in place of oatmeal. Bread also in different forms may be given. The crust of French bread is easily digested, and is supposed to have less starch in proportion to its gluten than the usual home-made bread. It is well to begin with some form of bread of this kind when we are getting the infant accustomed to take starch in the form of bread. If it is constipated, Graham bread and preparations of rye will also be found useful. Fresh bread should never be given, and bread one day old is the preferable form which should be provided.

The infant should never be given cake or candy even to taste. It is necessary to state this very decidedly, because it is an erroneous view which is held by most mothers that it can do no harm to give occasionally to an infant in its second year of life, or to a young child, a little candy or a little cake. This may be true so far as the immediate effect these articles may have on the digestion is concerned, but it is of far more importance that the infant should not have its taste perverted from those articles of diet which are best for its nutrition. These new articles appeal more strongly to its sense of taste, and allow it to know that there is something which tastes more agreeable than the food which it is accustomed to have. When an infant has acquired a taste for cake or candy, it will cease to enjoy the food by which its development will be best perfected. It is, in fact, kinder to the infant never to allow it to taste cake or candy. When these articles are withheld, it will continue to have a healthy appetite and taste for necessary and proper articles of food.

Broths can be prepared in the following manner:

CHICKEN BROTH AND JELLY.—A fowl weighing about three pounds, with two tablespoonfuls of rice, two quarts of cold water, and salt and pepper, should be boiled for two hours and until the fluid has boiled down to one quart. The fluid should be strained while hot through a fine sieve. If a jelly is to be made, the broth made in the above manner should be allowed to cool in an earthen jar for about twelve hours in the ice-chest. The resulting jelly can be used in full strength or diluted with water. When the jelly has been thoroughly cooled, the fat can be partially or entirely removed from the top.

MUTTON BROTH.—A shoulder of lamb, when it can be obtained,—otherwise of mutton,—weighing from five to seven pounds, is treated in the same way as is the fowl for the preparation of chicken broth.

THIRD NUTRITIVE PERIOD.

The third nutritive period I have arbitrarily made to begin at about the thirtieth month of life.

At this time it will be well to begin to accustom the child's digestive functions to a still greater variety of food. In summer the more easily digestible vegetables, such as squash, young peas, and young beans, can be given. The variety of fruits can also be increased at this period, but they should be cooked. The principal change which is to be made in the diet to which the infant has been accustomed is a very decided increase in the proportion of the proteid element of its food. This is accomplished by means of giving the child meat. The quantity of meat which should be given towards the end of the third year should be small at first, and should be given at intervals of a day or two. Meat as a regular article of diet for each day is not, as a rule, required until the child is between three and four years old. The kinds of meat which should be given in this early period of childhood are chicken, mutton-chop, roast beef, and beefsteak. These meats should be cut into small pieces, and a little salt added according to the child's taste. It is well during the third year to give the child an egg on one day and meat on the next.

When the child has reached the age of five or six years, we should allow it to have a somewhat more varied diet, but during the whole period of childhood up to the age of puberty the closest attention should be given to the regulation of the kind and the amount of food to be given to the child, and any deviations from the rules which have just been laid down are to be deprecated.

DIVISION III.

GENERAL PRINCIPLES OF EXAMINATION AND TREATMENT.

Method of examining a Child.—When a physician is called to see a sick child, he must, if possible, ascertain before entering the nursery what is the temperament of the child with whom he will have to deal, and by the aid of this information regulate the manner in which he approaches it.

An infant in the early months of life too young to fear a stranger, a child of quiet phlegmatic temperament, or one that is too sick to object to being handled, can be examined as soon as it is seen, with the regularity and precision which one would employ with the adult.

It is an entirely different task, however, when one is called upon to examine children who are nervous, excitable, or timid, or who are spoiled and vicious. In dealing with the first and more difficult class of these cases much deliberation in the way in which the child is approached is needed, and much tact in speaking to it is required. In the second, the spoiled and vicious class, time cannot be gained by delaying the examination, and the sooner it is made with firmness and persistence the less trying it will be for the child and for the mother. As a rule, the more the child cries and resists needlessly, the less likely is it to have any disease of serious import.

It is wise at first to make the child think that no notice is being taken of it. It is well to notice its toys, and to appear to take great interest in them and also in the pictures in the nursery. The child very soon will become accustomed to the physician's presence, and will begin to take the same interest in him that he seems to take in its toys. A nervous, timid child will often from this point of the examination allow itself to be examined without further trouble.

The physician, however, should always be gentle both in his voice and in his touch, and on the slightest appearance of timidity, or manifestation of a desire to avoid him, he should at once stop the special part of the examination which he is making, and appear not to take any notice of the child.

All these preliminaries and minute details, which seemingly delay the examination, in fact expedite it, since, when once the timid child is thoroughly frightened, the rest of the examination becomes very unsatisfactory, for it is almost cruel in cases of this kind to attempt to force an examination, which in the case of the vicious child can be done usually without cruelty and without hurting the feelings of the mother.

The faculty of examining the child when it is crying and excited with the same precision as when it is quiescent, though perhaps by a somewhat different method, should be acquired. The trained hand and ear can detect an abdominal or pleuritic effusion or a solidified lung almost as well when the child is screaming as when it is perfectly docile.

This is an accomplishment which should be mastered at once by every practitioner who expects to have children under his care. In fact, if this were more universally understood, we should hear less of the impossibility of determining what is the matter with a child on account of its being fractious.

As the physical examination of a child is more difficult than that of an adult, and needs to be made more quickly, every means should be employed which will tend to throw light on the final result.

HISTORY.—A complete history of the case is very valuable, and should be obtained from the mother and the nurse, preferably before seeing the child, for in this way the physician can obviate asking many questions in its presence, a procedure which frequently fatigues it and renders it more difficult to examine. It is well to allow the mother and the nurse to tell you in their own language what they know about the child and its sickness. After they have finished, you can easily systematize the history of the case by any questions which you may wish to ask. Although the history given by the mother and the nurse is usually imperfect and disconnected, yet it is very likely to supply certain important points which you in your questions might easily overlook. The mother and the nurse are so constantly with the child that they notice all the slight shades of difference in its condition from hour to hour, a knowledge of which is of great importance in obtaining a correct appreciation of the general condition of the child, whatever the disease may be.

The information especially to be elicited is in regard to the health of the parents, whether there is an hereditary tendency in the family; whether the mother has had miscarriages (in reference to a possible syphilis); whether there are other children, and, if so, the state of their health; if any have died, of what did they die; the birth weight of this child; was it nursed, and, if so, for how long; did it have a substitute food, and, if so, of what kind? Did it gain in weight regularly, and what is its present weight? At what age did it cut its first tooth, and how many has it now? At what age did it sit, and stand, and walk alone? What diseases has it had? How long has it been sick? What were the first symptoms? Its appetite? Its sleep? Its temper? The number of its movements in twenty-four hours? The physician should personally inspect the fæces, and should, if possible, obtain and examine a specimen of the urine.

Having now systematized in his own mind the history of the case, the physician on entering the nursery should proceed with his inspection of the child.

TEMPERATURE.—It is so important to ascertain what the temperature of the child is that, if possible, the temperature should be taken before the child has become frightened or fractious. The place for taking the temperature should be in the axilla or rectum. The most successful method of obtaining it is to explain to the mother and nurse exactly what you wish to have done. They should be directed to take the thermometer and show it to the child as though it were a toy, to put it under the child's arm, and to play with the child until they are told to remove the thermometer. A rectal temperature is always desirable when it can be obtained, as it is more exact, and is usually at least one degree higher than under the arm. As a rule, the temperature cannot be safely taken in the mouth in young children.

INSPECTION.—One of the most valuable means of making a diagnosis of disease in children is the careful inspection of the child before attempting to percuss or to auscult it. In fact, when children are irritable and restless the inspection becomes of the utmost importance, and an eye which has been trained to understand the different aspects of disease in children readily makes the diagnosis in many cases without further assistance. A rule to be remembered, and one which will be found of great practical value, is, if possible, to have the child entirely undressed, so that we may see the whole surface of the skin in front and behind. Not only shall we thus be able to recognize the symptoms attributable to a simple irritation of the skin, whereas otherwise we might be led to consider them as representing a more general and constitutional disturbance, but the skin will also be found to be a valuable index by which to judge of diseases of the other organs. The cyanosis which so frequently represents some disturbance in the heart or lung, the quick respirations of either a thoracic or an abdominal type, a sunken or a distended abdomen, and the position of the child, all point towards symptoms belonging to special diseases. By means of all these symptoms, which can be seen at a glance, the diagnosis of the special disease can, after considerable experience, usually be made without much aid from other sources.

RESPIRATION.—Either when the thermometer is under the child's arm or when the regular inspection is begun, the rate and rhythm of the respiration can be determined. Having determined the temperature and respiration, if all that is necessary about the child when it is quiescent has been seen, the physical examination should next be made.

PALPATION.—Palpation is a very valuable means of diagnosticating disease in children, whether it be of the abdomen or of the thorax. It is well to begin with an attempt to take the pulse. Sometimes this can be readily accomplished. At other times it is impossible, and, as a rule, I rely less on the rapidity of the pulse in the child than on the information which is received from the temperature and respiration. It takes so little to increase the rate of the pulse in a young child that if we were to judge in every case by it we should often be misled in our diagnosis. What we wish especially to learn is whether there is a slow pulse or whether it intermits. This we can usually ascertain by keeping our finger for a few seconds on the child's radial artery. When we have once obtained a fair idea of the rate and rhythm of the pulse we can proceed with the remainder of our examination by palpation.

A young child's thoracic walls are so thin, and vibration is so pronounced in them, that often we can detect what process is going on in the lung by merely putting our hand on the chest, and we can feel in a chronic bronchitis what will prove on auscultation to be coarse sonorous râles. We can also sometimes feel a pleuritic or a pericardial friction-rub, and frequently a roughening of one of the valves of the heart. It is not altogether impossible in certain cases to distinguish the difference presented to the hand between a pleuritic effusion and a solidified lung. The examination of the abdomen, even when the child is crying, can be accomplished with considerable precision. Waiting until the child stops crying for a second and relaxes its abdominal walls, by firm but gentle pressure the abdominal walls can be depressed so as to obtain a fair knowledge of whether an abdominal tumor is present. Fluid in the abdominal cavity can also readily be detected by palpation.

A rectal examination is often important in infants and young children. It can readily be made without hurting the child, and the finger is able to reach much farther proportionately into the child's pelvis than into that of the adult, and very much more can be learned by this method than in adult cases. An invagination or an appendicitis can be diagnosticated by the combined examination through the rectum and by external pressure when external palpation alone has failed to give evidence of disease.

In the infant the head should be carefully examined in reference to the fontanelles. Measurements should be taken of the head and of the thorax.

At this stage of the examination we shall have determined almost always what disease is affecting the child, but every known method should, of course, be made use of for verifying the diagnosis. We should, therefore, endeavor to percuss and auscult the child, but in a somewhat different way from that which would naturally be employed in the adult. The louder the child cries, the easier is it to obtain evidence, through vocal fremitus, of the nature of the disturbance in the chest.

PERCUSSION.—Even when the child is crying and resisting, percussion may be of the greatest importance. Light percussion, as a rule, is preferable to the deeper and heavier percussion. The chest walls are so resonant that deep percussion rather masks the process which is directly under the finger by bringing out sounds from all parts of the chest. Direct percussion with the finger is preferable to the use of any instrument, as in this way both palpation and percussion may be combined. Palpatory percussion in my hands has always proved exceedingly valuable for purposes of diagnosis. A few light taps over the normal boundaries of the heart and lung will give much information, even though it may be impossible to obtain a more extended percussion of the chest. If the child is crying, one should watch until it takes its breath; just as it inspires it necessarily stops crying, and at that moment a perfectly clear percussion note can be obtained.

Care should be taken not to make the physical examination too protracted. Rapidity, both in palpation and in percussion, is very important, and a young child should be examined with much greater rapidity than is usual or necessary in the case of an adult. Much more information will be obtained in this way than if the child is wearied by continual efforts to make sure that no mistake has been made.

The sounds which can be elicited from a young child's chest are so varied that it is more difficult to differentiate them than in the adult. If one hesitates and doubts one will not arrive at as correct a result in the examination as when the mind is trained to grasp at once the salient points in the special physical examination, and to depend somewhat more on the first idea which is formed than would be wise in the older cases.

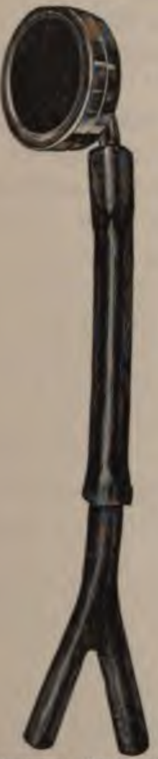
AUSCULTATION.—I have noticed that children are much more sensitive to the feeling of the stethoscope than are adults. In many cases they shrink from it as though it hurt them, even when they have not been frightened by the previous part of the examination with palpation and percussion. It is, therefore, exceedingly important to make the examination as pleasant to the child as possible. A rubber cup applied to the end of the stethoscope serves this purpose well. The feeling of the soft rubber is pleasant to the child, and it conveys the sound with almost as much clearness as does the hard rubber end of the stethoscope. The stethoscope should have flexible rubber arms so as to follow easily the motions of the child, and its bell should be of such small caliber that it can easily be introduced between the ribs of even a young child.

In like manner a phonendoscope with a small disk, such as is represented in Fig. 58 is valuable where the sounds of the lungs or heart are very feeble, or when it is found preferable to examine without having all the clothes removed.

It is, however, often of great aid in the proper appreciation of the sounds which are heard with the stethoscope in infants and in young children, especially when they are crying, to use a stethoscope which does not convey the sound so clearly and intensely as do others. We can often in this way differentiate a soft cardiac murmur which if a more delicate instrument were used would be entirely obscured by the loud sounds coming from the trachea and bronchi of a crying or screaming child. We can, also, often distinguish the fine râles of a broncho-pneumonia in contradistinction to the loud coarse râles which tend to obscure the other sounds in the chest.

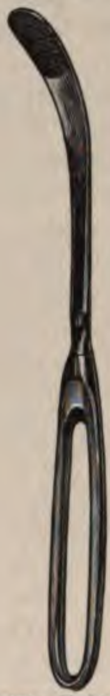
EXAMINATION OF THE THROAT.—The examination of the throat should be left until we have practically finished with the general examination of the child, because, as a rule, it is the procedure of all others which irritates it, and after we have once attempted to examine the throat we shall seldom be forgiven by the child at that special visit. Some children will allow you to look into their throats without being at all disturbed. As a rule, however, it frightens them, and we should use the most gentle and rapid methods for accomplishing our purpose. We must not expect to be able to sit down in front of the child and examine its throat for some minutes, as is possible with adults. We must adopt some definite method by which we can control the child and catch a glimpse of the mouth, tongue, and pharynx. The more quickly we do this, the less it frightens the child, and it is important that we should not make extensive preparations which it will notice and which will indicate what we are going to do. The mothers are often much disturbed by seeing the child first frightened with the idea that it is going to have a spoon put in its mouth, and then, while screaming and crying, forced to the window and compelled to open

FIG. 58.



Phonendoscope. Reduced two-fifths.

FIG. 59.



Tongue depressor. Reduced 56 per cent.

its mouth. It is far better under all circumstances to tell the mother and the nurse what to do, and for us not to go near the child until they are entirely prepared to control its limbs and are holding it in a position in which it is practically helpless. It frightens the child much less to have it sit in the nurse's lap with its face to the window than to examine it on its back.

The nurse should hold the child firmly against her chest, so that it cannot move its arms or legs or slip from her lap. The physician should control the child's head with one hand while with the other he gently introduces the handle of a spoon into the mouth, passing it back until it touches the soft palate, when the child will gag, and a quick glance can be obtained of the tonsils and pharynx.

I prefer to use a spoon for examining the throat, because in every household one may be easily obtained, and it obviates the use of the same instrument in a number of mouths, which is something to be considered in children, in whom infection by the mouth is so common. Of course, for those who prefer to use the usual tongue-depressor the danger is reduced to a minimum if a careful disinfection of the instrument is made after it is used; but in the case of infants, who should also be examined in an upright position, the spoon is decidedly preferable, because the neck of the infant is so short that its chin is in close proximity to its chest, and the handle of the tongue-depressor interferes with the proper downward pressure of the instrument. The spoon-handle, on the other hand, is exactly the shape which is best adapted to the infant's mouth and tongue, and the spoon, being comparatively straight, does not encroach upon the thorax when the downward pressure is made.

Fig. 59 represents a tongue-depressor devised by Dr. Henry Chapin, of New York, and is remarkably well suited for the examination of the infant's throat, as the curve is adapted to that of the tongue.

THE THROAT.—The throat of the child may often be affected, and be the only source of the symptoms, although these symptoms may not be what one would expect to find associated with a lesion in this region. Young children are so apt not to complain of their throat, and to show merely signs of general constitutional disturbance, that the physician is very likely to be misled and to overlook the real seat of the disease unless he makes it a rule always to examine the throat at his first visit and in all obscure cases.

INSPECTION OF THE MOUTH.—It is well when the physician is examining the throat of an infant in the first two years of its life, and even later if there are any symptoms which point towards the mouth, to examine the gums carefully. The hands should be carefully washed before introducing the fingers into the mouth. This is in accordance with the common rules of cleanliness, and also is required in order that the introduction of pathogenic organisms into the infant's mouth should be avoided. In examining the gums we judge whether they are swollen or reddened, dry, moist, or hotter than normal, and also whether there is a condition of the gums which indicates the use of the lancet.

Examination of the Ears.—One of the most important means of rightly interpreting the symptoms of restlessness, of evident pain, of heightened temperature, of undue somnolence, as well as a great many other symptoms, is the examination of the ears of infants and of young children. A slight irritation in the throat may at times cause a congestion in the vessels of the membrana tympani which may produce all these symptoms.

It is, therefore, very important, unless you are sure that the symptoms do not arise from some condition in the ear, that you should examine the ears at some time during your visit, choosing that time which seems most favorable in the especial case. A thorough knowledge of the possible symptoms which may arise from the ear is of the very greatest importance for the general practitioner to possess.

Lumbar Puncture.—Paracentesis of the vertebral canal is of great aid in certain cases. This procedure is employed for diagnosis in obscure cerebral disease, and enables us to determine, first, whether a meningitis is present, and second, whether it is a form of tubercular, non-tubercular, or cerebro-spinal meningitis.

Röntgen Light.—The X-ray has been used more extensively and successfully in surgical cases than in medical, and its use for medical cases in children has so far been rather limited. As in diseases of the heart and lungs it is not only their size but their movements which we wish to observe, the fluoroscope is better adapted for detecting pathological conditions. Examination with this instrument can also be made more quickly than with the radiograph, which requires more time to develop, but gives more details of those organs which are at rest. An example of the extent to which radiography is applicable for showing the outlines of the different organs, even when they are moving, can be seen in Plate IV., facing page 254, which represents the thorax and abdomen, with their contents, of a child of six months.

It is now claimed that with the fluoroscope thoracic and pericardial effusions, even when small, can be detected; also that a central pneumonia can be recognized before there are any signs on auscultation and percussion, and that the area of consolidation of a pulmonary tuberculosis can be diagnosticated by the diseased area of the lung appearing darker than normal, and by the restriction of the excursion of the diaphragm in quiet breathing and in full inspiration. An enlarged heart or a transposed heart can also be detected with the fluoroscope. The X-ray photograph reveals changes in the bones, such as occur in rhachitis (Plate V., facing page 336); in tuberculosis (Plate VII., facing page 512); in osteomyelitis (Plate VII., facing page 512); in syphilis (Plate V., facing page 336); and also changes in the various abdominal organs. Radiography is therefore probably destined to become a very important aid in our diagnosis in the diseases of children, and should always, if possible, be used in obscure cases, such as we are continually meeting with, especially in the lung in which a central pneumonia may be masked for many days, and yet the child be so ill as to cause the most serious apprehensions as to diagnosis and prognosis.

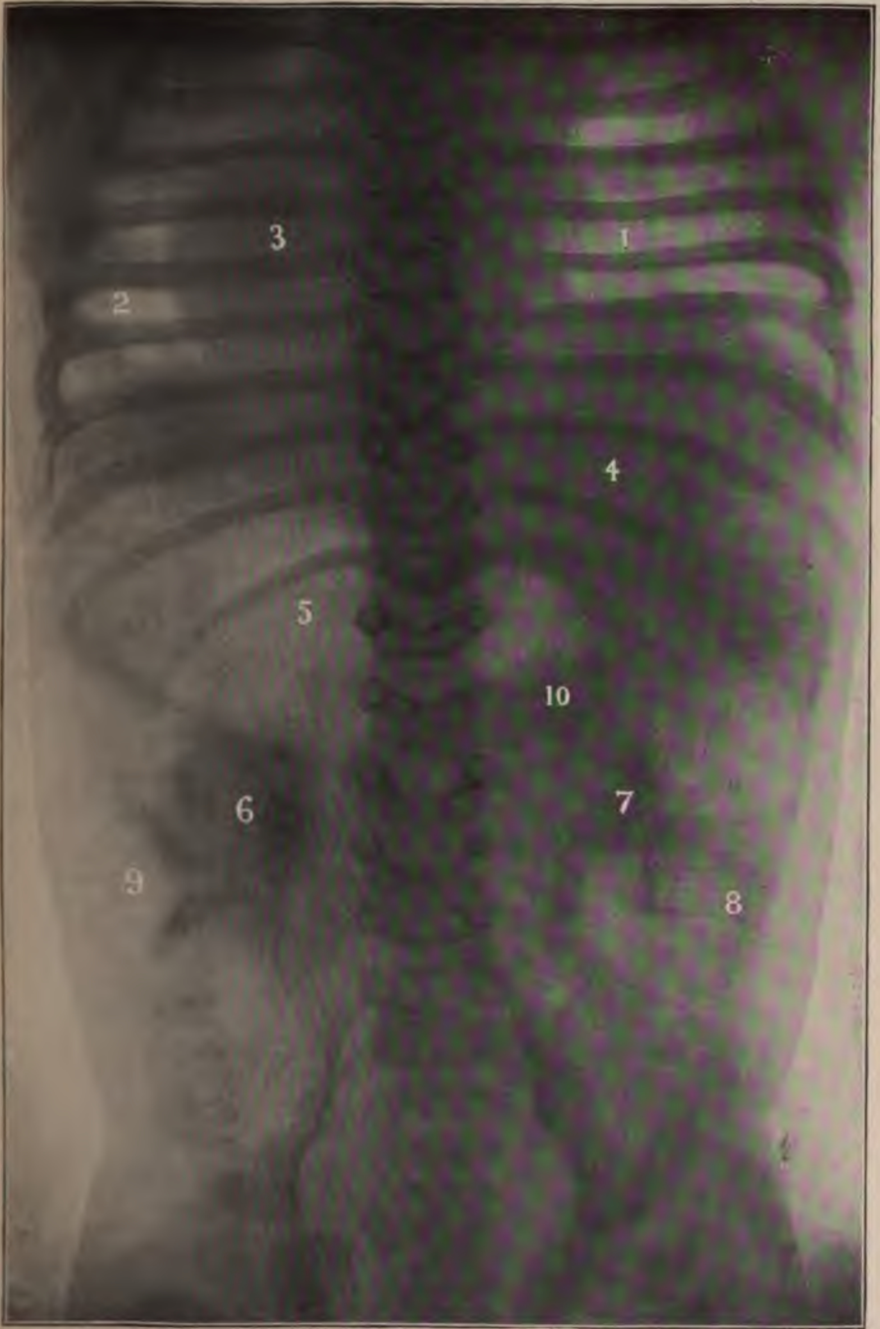
TREATMENT IN GENERAL.—The treatment of infants and children consists largely in the administration of a *food* adapted to the condition of the child, and in gradually changing from a weak food to a strong one, or *vice versa*, according to the especial indications in the disease as it runs its course. Of great importance also is the regulation of the *temperature* and the *purity of the air* in the sick-room. In many cases, especially in the first year of life, a change from one room to another several times during the day is advantageous, and the room should be as large as possible. The application of *cold* and *heat* by sponging and baths, and the technique of *gavage*, *lavage*, and *intestinal irrigation*, will be spoken of in describing the treatment of the various diseases in which they are indicated.

Stimulants, if used with good judgment and given as described in the treatment of the various diseases, are very valuable, and are called for not only in cases of sudden collapse, in which their administration by subcutaneous injection is indicated, but whenever there is depression with a weak or intermittent pulse, as in diphtheria, at times in typhoid fever, and also where there is a sudden fall in temperature, as is frequently seen at the crisis of a pneumonia. The amount of the stimulant for the especial age cannot be given, as it depends entirely on the disease and the condition of the especial patient. The form of stimulant which is borne best by young children is brandy or whiskey. The contra-indications for stimulants are found in those cases in which the child has a hot, dry skin, flushed face, and a full, regular, and tense pulse. Great caution should be used in the administration of stimulants, as there is no doubt that they are given with but little judgment in many cases, and that to obtain the best results they should be reserved for cases in which they are clearly indicated. They should be given in rather small doses, the larger doses being reserved for special conditions. As a rule, the average practitioner gives much larger doses of stimulants than are needed.

Especial attention should be drawn to the fact that the younger the infant the greater is the importance of keeping the nasal passages clear, irrespective of the disease. The infant responds much more quickly to treatment if its vitality is not lowered by its efforts to breathe through its mouth rather than through the natural passages. A spray of oil is the treatment in young children for occluded nares. *Nasal irrigation* is described under the treatment of diphtheria on page 473; *enemata* on page 830, and *atomization* of the throat and larynx on page 655.

An important fact to remember in the treatment of infants and young children is that *drugs* play a very insignificant part in the actual cure of diseases. According to my observation, numbers of children are being treated by drugs, and yet often, so far as I can see, this time-honored means of satisfying parental prejudices is but prolonging the symptoms of

PLATE IV.



Infant, 6 months old. Taken under chloroform from behind. Stomach and large intestine distended with gas. 1, right lung; 2, left lung; 3, heart; 4, liver; 5, stomach; 6, left kidney; 7, right kidney; 8, ascending colon; 9, descending colon; 10, probably the head of the pancreas.

a disease which, self-limited, has run its course. I do not for a moment question the direct benefit obtained from quinine in malaria and mercury in syphilis: it is the promiscuous use of drugs in every case of sickness to which I am especially opposed, for in many cases the child will recover with equal or even greater rapidity without them.

Instances probably arise in the practice of every physician in which he feels that the drugs which have been given have either directly harmed the child or, by disturbing its digestion and thus interfering with its nutrition, have indirectly produced more serious symptoms than those presented by the original disease. The greatest caution should be employed when drugs are used with young children, and there should be a thorough understanding of their action during the various periods of development. The well-known susceptibilty of children to the action of opium and its alkaloids should make us careful to begin with minimum doses when it is necessary to use this drug. In like manner, although it is traditional that children have a great tolerance for belladonna and arsenic, we must allow that an overdose of the former, although not usually fatal, may certainly produce most alarming symptoms, while the administration of the latter as I have seen it given in the treatment of chorea has in a number of cases produced a multiple neuritis.

The treatment of diseases by special drugs because these drugs have been given in the past, because their administration has apparently done no harm, or because no new or better remedy has been found, rests upon a lack of comprehension of what treatment really means.

The custom of combining many drugs in one prescription is fallacious, and should be discountenanced, especially when infants and young children are being treated. A single drug given in the smallest dose which will accomplish its purpose, and in the most agreeable form which is compatible with the function of digestion, will produce the best results in any given disease.

The delicate skin of infants and young children is peculiarly sensitive to reflex disturbances caused by drugs in the gastro-enteric tract, and therefore we must be careful not to mistake the appearances produced by such reflex irritation for the various lesions of the skin which may occur in a specific disease. Thus, the similarity of the efflorescence produced by belladonna to that accompanying scarlet fever is striking. Almost any drug, as well as certain articles of diet, may in some individuals produce forms of papular erythema, resembling very closely some of the dermal lesions of syphilis. It is therefore wise to avoid these possible disturbances of nutrition by giving drugs only when they are actually known to be necessary, and by omitting them as soon as possible.

It has always seemed to me irrational to prescribe syrups as a menstruum for the administration of drugs to children. Their well-known tendency to fermentation is sufficient to stamp them as unfit for the treatment of a period of life when the undeveloped condition of the digestive function indicates the vital importance of protecting this function in every way.

Each case must be treated according to its special pathological lesion or specific micro-organism. As year by year we are discovering the organisms which cause special diseases, so the treatment of the future will be the actual destruction and speedy elimination of these organisms while supporting the strength until such elimination has been accomplished. When no known organisms exist, the treatment should be if possible to remove the cause, and to support the vitality until natural processes have healed the special lesion, produced either by exposure or by trauma.

PROPHYLAXIS.—At no period of life is the prophylaxis of disease so important and its results so brilliant as in infancy and early childhood. When physicians have sufficiently recognized the fact that disease in young children results in the vast majority of cases from a disregard of the laws of hygiene and of rational methods of feeding, they will so impress upon the laity the true meaning of infantile prophylaxis that the mortality rate will be greatly reduced.

DIVISION IV.

PREMATURE INFANTS.

A PREMATURE infant is one which is born prior to the usual two hundred and eighty days which represent the normal duration of intra-uterine life.

Very few cases are reported, and none of them appear to be absolutely authentic, in which an infant has survived which was born much before the twenty-seventh or twenty-eighth week of intra-uterine life. The premature infant in its intra-uterine development is unprepared to meet the conditions of extra-uterine life, and often dies within a few days, and usually within a few hours.

DETERMINATION OF AGE.—A sufficient number of careful investigations regarding the characteristic appearances and the development of the fœtus during the last four months of intra-uterine life has not yet been made and recorded to enable us to state definitely what age the infant represents when it is born. The few facts which we possess concerning this subject must, however, be made use of, and, though not absolutely correct, are sufficiently so to be of great value to us in our management of these cases. One reason for the difficulty which arises in every case in determining the age of the fœtus is that the conditions which influence its growth during intra-uterine life are very varied. The health of the mother and her hygienic surroundings, together with the influence of heredity on the size of her offspring, present good reasons for decided variations in the growth of the fœtus in different cases at the same period of intra-uterine life.

If the infant is living when it is born, we should at once carry out the rules for preserving its life which have proved to be best in the case of any infant born prematurely. These rules should be insisted on even if the infant has been born at a much earlier stage of development than is, according to our present ideas, compatible with its viability. This is necessary, because so many errors in our calculation as to when the impregnation took place are liable to arise, and also because a fœtus may have arrived at a period of intra-uterine development which is perfectly compatible with life, and yet from its small weight and general characteristics have the appearance of one whose development is incompatible. Whatever advances we may make in the future in preserving the lives of premature infants born at an earlier date than is supposed to be compatible with life,—namely, from the twenty-fourth to the twenty-eighth week,—it would hardly be practical at this time to discuss the treatment of infants born before the twenty-fourth week.

AT TWENTY-FOUR WEEKS.—A fœtus born at about the twenty-fourth week of intra-uterine life usually breathes feebly, and dies in the course of a few hours, apparently from an inability to accommodate itself to conditions for which it is not prepared. At this stage of development it may still have fine hair (lanugo) over the whole of its body, but it is often the case that this hair, commonly found from the sixteenth to the twentieth week has disappeared. At this age it still has very little deposition of fat in the subcutaneous cellular tissue, and it has a decidedly emaciated appearance. In other respects, except in size, it does not differ very much in its appearance from the fœtus of some weeks' later development. Its eyelids have separated, though it is so feeble that, as a rule, it cannot open and shut them.

The estimation of the length of the fœtus is difficult to make, and, on the whole, unsatisfactory and inexact. These measurements, in all probability, differ very much when made by different investigators, owing, as Minot has pointed out, to the many changes in the curvature of the longitudinal axis of the human embryo, which make it impracticable to employ any one system of measurement in obtaining comparable results for all ages. Hecker's figures, however, are probably as reliable as any we know of. According to this author, at about the twenty-fourth week the fœtus measures 28 to 34 cm. (11¼ to 13½ inches). Its weight, according to Lusk, is about 690 grammes (23 ounces).

AT TWENTY-EIGHT WEEKS.—By the time the fœtus has reached the twenty-eighth to the twenty-ninth week of intra-uterine existence its condition, so far as its development is concerned, is such that there is no necessary contra-indication to its living if it happens to be born at this time. It has been stated that an infant born prematurely at the twenty-eighth week is more likely to live than one which is born at the thirty-second week of intra-uterine life, and that this has been proved by statistics. If true, the reason for this, I believe, is because much greater care is taken of the former than of the latter. It is reasonable to believe that an earlier stage of intra-uterine development is less likely to insure continuance of life after premature birth than a later stage, provided the same precautions are taken in each case.

Hecker's and Lusk's figures, in a general way, state that when the fœtus is born at about the twenty-eighth to the twenty-ninth week it measures from 35 to 38 cm. (about 13¾ to 15 inches) and weighs about 1170 grammes (39 ounces). The skin is still wrinkled, is of a dull red color, is covered with vernix caseosa, and there is very little deposition of subcutaneous fat. The infant can move its limbs slightly, cries feebly, and often dies in a few hours or days.

AT THIRTY-TWO WEEKS.—Again, using Hecker's and Lusk's figures for the thirty-second, thirty-sixth, and thirty-eighth weeks, at about the thirty-second week of intra-uterine life the fœtus measures from 39 to 41 cm. (about 15½ to 16½ inches) and weighs about 1560 grammes (52 ounces). The hair of the head by this time has increased in thickness, and the lanugo, which in many cases is pronounced from the twenty-eighth to the thirty-second week, has either begun to disappear or has entirely disappeared from the face. The nails, which between the twenty-eighth and thirty-second weeks are often not well developed, now present a normal appearance, though they frequently do not quite reach the tips of the fingers. At this age, also, in boys, it is often possible to feel the testicle in the scrotum. There is usually, also, in a healthy fœtus, considerable deposition of subcutaneous fat, and the senile aspect of the earlier periods of intra-uterine life is much lessened.

AT THIRTY-SIX WEEKS.—At about the thirty-sixth week the length of the fœtus is from 42 to 44 cm. (about 16¾ to 17½ inches) and its weight is about 1920 grammes (64 ounces). The lanugo has usually at this period disappeared, and the infant, although less energetic than at full term, is decidedly stronger than in the previous periods which have been mentioned. It sleeps a great deal, and is still in a condition to die easily unless carefully looked after.

AT THIRTY-EIGHT WEEKS.—At about the thirty-eighth week of intra-uterine life the infant measures about 45 to 47 cm. (about 17¾ to 18¾ inches) and weighs about 2310 grammes (77 ounces).

NORMAL DEVELOPMENT.—There have been so few observations recorded of the development of the various parts of the fœtus in the latter months of intra-uterine life that I am not prepared to describe systematically the development of the premature infant as I have already done that of the infant at term. There are, however, certain facts which I have observed and others which have been recorded.

HEAD, THORAX, AND ABDOMEN.—All those anatomical conditions which have been emphasized in describing the infant at term as being especially prominent are still more marked in the premature infant. Thus, the head is large in comparison with the thorax, and the abdomen is in almost every case much distended in premature infants, owing to the large proportionate size of the liver. This distention of the abdomen lasts for many weeks, and even months, and its gradual return to the normal size and appearance is one of the signs that the infant is doing well and is gradually acquiring the normal anatomical development of the infant born at term.

SKIN.—The various changes in the color of the skin, already described as represented by erythema neonatorum and icterus neonatorum, I have noticed to occur in the premature infant as they do in the infant at term.

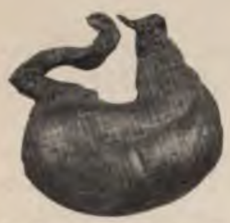
SWEAT-GLANDS.—The function of the sweat-glands is, as a rule, not developed at birth, and we do not expect the premature infant in the early weeks of life to perspire. There is, however, a great variation as to the time of the development of the function of the sweat-glands. In an infant premature at seven and one-half months I have noticed free perspiration take place after it had been born one week.

FEET.—Some dissections made by Dane on the feet of an infant premature at the seventh month show that the foot at this stage of development closely approaches in external appearances the well-developed foot of the adult. The dissections also show a remarkably well-constructed bony framework.

GASTRIC CAPACITY.—As the question of the proper amount of food to be given to a premature infant is of the utmost importance, it is well to know about what the average gastric capacity of the fœtus is during the later months of intra-uterine life. No series of complete and reliable observations on this point have been made, that I know of, and the rules by which we are guided must for the present be very general ones. The less the weight of the infant, the less, in many cases, is the gastric capacity.

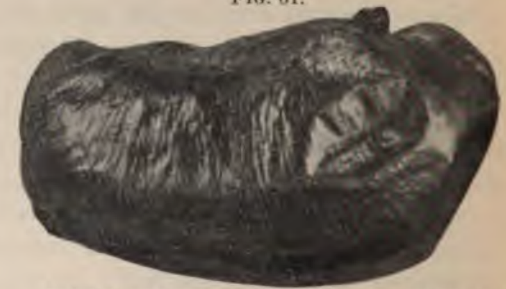
The following figures represent the fœtal stomach at four and one-half, seven and one-half, and eight months. Fig. 60 represents the stomach at four and one-half months, and is interesting merely as showing the relatively advanced development of the lesser and greater curvatures at this age, as well as the rapid growth which takes place between the fourth and the seventh month.

FIG. 60.



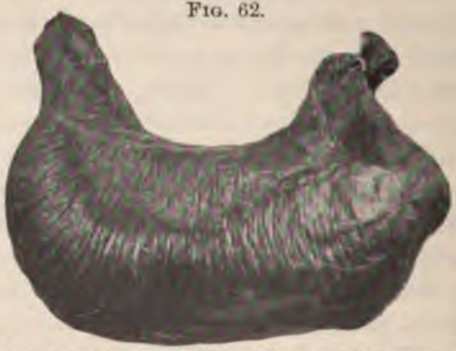
Fœtal stomach (natural size), 4½ months old.

FIG. 61.



Fœtal stomach (natural size), 7½ months old. Weight of fœtus, 1920 grammes. Gastric capacity, 18 c.c.

FIG. 62.

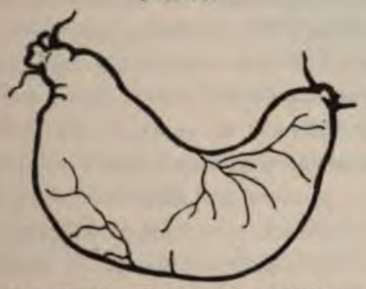


Fœtal stomach (natural size), 8 months old. Weight of fœtus, 1230 grammes. Gastric capacity, 22 c.c.

The next stomach (Fig. 61) was taken from an infant born prematurely at about the twenty-ninth to the thirtieth week. It is of rather peculiar shape. The weight of this fœtus was 1920 grammes (about 4 pounds). Its gastric capacity was 18 c.c. (about 4½ drachms).

The next stomach (Fig. 62) was taken at about the thirty-second week from a fœtus which died in forty-five minutes from the time of its birth. The gastric capacity was 22 c.c. (5½ drachms). The weight of this infant was 1230 grammes (2 pounds 9 ounces).

FIG. 63.



Fœtal stomach (natural size), 8 months old. Weight of fœtus, 1440 grammes. Gastric capacity, 8 c.c.

The next stomach (Fig. 63) was taken from a fœtus born at about the thirty-second week of intra-uterine life, and weighing 1440 grammes (about 3 pounds). Its gastric capacity was 8 c.c. (about 2 drachms).

Intestinal Contents.—The meconium in premature infants presents the same appearance as is seen in infants at term. When the food is properly regulated, the fæcal discharges assume the consistency and color which are seen in those of infants who have been born at term. This color in its usual varieties is well represented in Plate III., 6, 7, 8, 9, facing page 84.

Amylolytic Function.—The amylolytic function of the infant at term is so slightly developed that we may safely assume that it should not be depended upon for the digestion of starch in the premature infant under any circumstances.

Sugar.—Although we must assume that the function of absorbing sugar is not developed to the same extent in the premature infant as in the infant at term, yet, in all probability, it is more highly developed than the other functions of digestion. Sugar is needed to keep up the animal heat of the premature infant, which is so very much more readily lessened than in the infant at term. Sugar, therefore, is an important element in the premature infant's food, but should be given at first in a much lower percentage than later, when the equilibrium of the gastro-enteric tract has been acquired.

Fat and Proteid Digestion.—The function of digesting fats and proteids is in a much more undeveloped condition in the premature infant than in the infant born at term, and should, therefore, not be depended upon to the same degree as can safely be done in arranging the food for the older infant. Much smaller percentages of these elements should be given to the premature infant than to the infant at term, both for purposes of digestion and of absorption, for, in all probability, the power of absorption of the gastro-enteric tract in premature infants is in a very undeveloped condition. As small a percentage of caseinogen in the total proteid as possible should be given.

KIDNEY.—We should expect, from the lack of development of the kidney in premature infants, to find a considerable deposit of uric acid, such as has already been described as appearing in the early days of life in infants at term. (Plate III., 1, facing page 84.) This is, in fact, the case, and the appearance of uric acid on the napkins of premature infants is, therefore, not necessarily to be looked upon as denoting an abnormal condition. It should, however, be carefully watched, for when it becomes excessive it is an indication that the infant's food has not been properly adjusted to its digestive powers and that the infant may soon begin to fail.

CIRCULATION.—The heart in premature infants has not yet arrived at the complete stage of development needed to render it a reliable central force which can fulfil the demands that will be made on it in the external world to sustain the equilibrium of the circulation. Therefore as little work as is possible should be thrown upon the heart, and the infant should be kept quiet, and not be carried about, as is customary with infants born at term. In a number of cases which I have carefully examined I have failed to detect a cardiac murmur, which leads me to think that the foramen ovale closes soon after birth in the same manner as it does in the infant at term.

ANIMAL HEAT.—The animal heat of the premature infant is much more easily reduced, and is even more important to its vitality, than it is in the infant at term. Following the rule that the smaller the size of the human being the greater proportionately is the entire surface, and, therefore, the greater the opportunity for lowering its temperature, an atmosphere which is suitable for the infant at term is too cold for the premature infant.

Premature infants should be thoroughly protected from changes of temperature of the atmosphere in which they live, and this temperature should be raised to a point which will correspond in some degree to that of intra-uterine life.

AIR.—Just as a necessity exists for the premature infant to live for some weeks in an atmosphere in which the air approaches in its temperature the warmth which exists in intra-uterine life, so is it almost to the same degree important that the air which it breathes should be free from dust and micro-organisms. The lung is in a very undeveloped condition, and although it may be sufficiently developed to carry on the function required of it in extra-uterine life, yet all its tissues are exceedingly sensitive, as are those of the nose and the naso-pharynx through which the air must be introduced to the lungs. The air of the ordinary room in which infants live when they are born necessarily contains many impurities, both irritating and morbid. This irritation of the respiratory passages may of itself be sufficient to reduce the vitality of the infant beyond the limits of life. In addition to this, as the mucous membranes of the premature infant are not fully developed, the infant is more vulnerable to the invasion of pathogenic organisms than at a later period.

TOUCH.—Premature infants have to be carefully handled, as they die easily from influences which would have little or no effect upon the infant born at term. In intra-uterine life they are floating in a fluid which practically prevents what in the external world corresponds to *handling*. While they are living in the amniotic fluid they are almost completely protected from the influence of touch, which necessarily affects them as soon as they are born. *Touch*, then, is an important element, to be avoided as much as possible when the premature infant is born, as it has a decided tendency to lower the vitality.

An instance of the care which is needed to preserve the lives of these infants came to my notice in the case of an infant premature at eight months which was in my service at the City Hospital.

During the first week or ten days of its life this infant was in charge of an unusually careful and experienced nurse, who appreciated the risk of handling it. It was gaining in weight and was doing well; but unfortunately another nurse was substituted who did not understand this class of infants so well. She allowed the patients in the ward to handle the infant, to talk to it, and to surround it with various similar deleterious influences. For a few days it lost in weight, and then it suddenly died. There is no doubt that it was unable to withstand the amount of handling, which would have done no harm to an older infant.

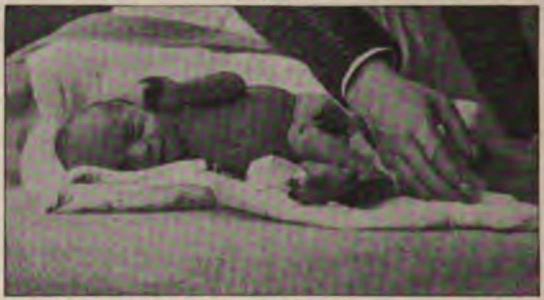
LIGHT.—The premature infant should live in comparative darkness during the early weeks of its life. Light is not requisite for the development of the infant in the earlier stages of its existence, and too much light will impair its vitality. It is important to adapt the light to the stage of its development, and gradually to accustom it to more light as it grows older.

SOUND.—In the normal intra-uterine conditions the infant is very slightly exposed to sound, and all its functions are adapted to silence rather than to the many noises which unavoidably surround it in the external world. We should therefore so arrange that from the minute it is born it is protected from noise.

PULSE, TEMPERATURE, AND RESPIRATION.—I have not very exact records of the average pulse, temperature, and respiration found in premature infants. These infants seem to present rather irregular types of temperature and pulse, as well as of respiration. They have to be so carefully handled that observations as to these physical signs must be made with great caution. The main point in regard to these three conditions of the premature infant is that they are all represented by irregularity. The *temperature* of the premature infant, when it has once begun to gain in weight and to thrive, is usually a little above the normal temperature of the infant at term. Before it has begun to gain in weight and when its vitality is much depressed, the temperature, as would naturally be expected, is rather below the normal standard; and we should watch this sign with the greatest solicitude, as a decided and continuous depression is often indicative of death. The *pulse* is difficult to take in the premature infant, and, as a rule, is somewhat quicker than in the infant at term. The *respirations*, irregular in the infant at term, are still more irregular in the premature infant, at times being rapid for a few seconds, and then becoming almost imperceptible for some minutes.

APPEARANCE AT BIRTH.—The picture of a premature infant in the early days of life is quite characteristic. Besides its very small size, as shown in Fig. 64, where the size is compared with the nurse's hand, it shows in varying degrees an absence of the life and vigor which is seen in the fully developed infant at term. It is emaciated, its skin being soft, wrinkled, and showing very little subcutaneous fat. Its head is large, its abdomen broad and distended, and its limbs puny. According to the stage of its development it may or may not have the remains of the hair (lanugo) on its body which was present in uterine life, and in like manner its nails may or may not be well formed. Its face has a senile expression and it is torpid and extremely somnolent. The eyes are closed. Its cry is very feeble. The surface temperature is usually cool, the extremities seldom move. The respirations are very superficial and irregular, often ceasing altogether for a few seconds. The power to suck and even to swallow is often slight. These signs evidently indicate that the vitality is very low, and if the weight is below three or four pounds and the length less than eighteen or nineteen inches, that the functions and organs are not developed sufficiently for use, and that unless unusual care is taken in the treatment of such cases, they will soon die.

FIG. 64.



Infant premature at seventh month. Birth-weight, 1740 grammes. Age, 10 days; weight, 1540 grammes.

TREATMENT.—The treatment of a premature infant should be begun at once, as every minute of the exposure to which infants at term are usually with safety submitted is of the greatest danger to the premature infant, and greatly enhances the difficulty of saving its life. The bodily temperature, on which the vitality is dependent, is reduced very rapidly, and exposure to such conditions as are abnormal to those of intra-uterine life and to undeveloped organs is to be at once guarded against. The abnormal conditions to be avoided are light, sound, touch, cold and impure air. To accomplish this the following rules are indicated:

I. There should be a receptacle which shall guard the infant from the deleterious influences of extra-uterine life.

II. The receptacle should be such that it can be obtained quickly and transported rapidly, and therefore should be kept at some central and convenient station.

III. The place where the receptacle is kept should be free from the influence of any disease.

IV. The receptacle should be so constructed as to make it possible for it to be absolutely cleansed and disinfected each time after it has been used, hence it should be made of metal.

V. The receptacle should, as soon as the infant is placed in it, be under the observation of trained nurses night and day.

VI. The food for the infant should be regulated with the greatest precision, with the closest attention to minute details, and if possible, at a milk-laboratory.

VII. The premature infant should not be bathed, but should be rapidly covered with warm, fresh sweet oil, and wrapped up in absorbent cotton, only the face being left exposed. The cotton around the buttocks should be separate from that around the body, head, and upper extremities, so that it can be changed oftener, as after the discharge of urine and fæces. The remaining cotton need not be changed oftener than once in forty-eight hours, and at each change of cotton the oil can be reapplied.

It will be found that the cotton is remarkably cleansing, and will obviate entirely the necessity for using water. The cotton and oil should be thoroughly warmed before being used. The infant should not be removed from its receptacle when these changes are being made or when it is fed. The surroundings and receptacle should be adapted to the indications just stated. All the possible causes which may reduce the vitality must be thoroughly understood and obviated, and it must be appreciated that a failure to recognize and obviate any one of these causes may defeat the benefit which should arise from attending to all the others. The premature infant, therefore, should, so far as possible, be restored to the condition that it has been forced out of,—namely, a condition of darkness, silence, warmth, and a medium free from physical shock and pathogenic micro-organisms.

Weight.—It is important to remember that the weight of premature infants of the same age varies at birth, just as we have seen in the case of infants born at term.

In treating these cases, observance of their weight is of the greatest importance, and until we have obtained a regular progressive daily increase in their weight we are never sure that they are thriving sufficiently to live. The daily gain which the premature infant should make has not yet been determined, but it is much less than is expected when an i is born at full term, and may be stated to be about 10 to 20 gramm to ⅔ ounce). Any decided loss in weight, such as 30 to 40 gramm to 1⅓ ounces), beyond what would occur from natural causes, sh make us look upon the infant as being in a critical condition and im upon us the importance of taking active measures to prevent further This loss in weight must, as it is relatively so small, be carefully adju to the loss which naturally occurs from the fæcal discharges. Thus, total amount of loss in weight from the fæcal discharges may amoun these premature infants to from 30 to 60 grammes (1 to 2 ounces) for fæcal discharge, and this may entail a considerable loss of the infa weight in the twenty-four hours beyond that occasioned by defec nutrition.

The knowledge of the daily weight of a premature infant is the p cipal index of the changes in its vitality which occur very rapidly, an is by the weight that we are guided in our daily adaptation of the food the infant's condition, and are also informed as to whether greater or l warmth, more oxygen, or stimulants are indicated.

The handling, however, which is usually necessary to obtain t weight is dangerous, as it reduces the vitality. This danger should obviated by having the receptacle balanced on correct and sensiti scales. The premature infant's life is so difficult to preserve that w should make use of every device which our ingenuity can suggest an which is within the limits of possibility of the especial case which we a called upon to treat. The receptacle for the immediate occupation of th newly born premature infant therefore becomes a very important pa of the treatment.

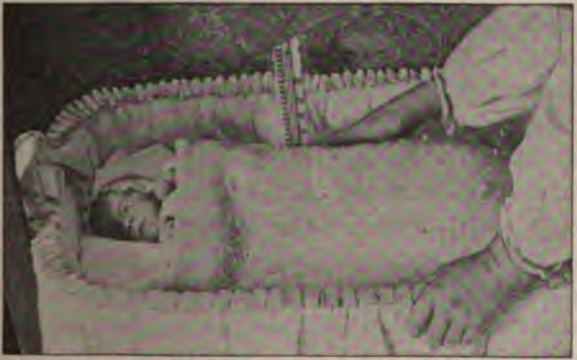
Receptacle.—In the treatment of premature infants only one of th principal methods of maintaining their viability usually receives much attention. It is commonly supposed that if the atmosphere which surrounds the infant is kept at a sufficiently high temperature all that is requisite has been done for its safety. This is sometimes accomplished by placing the infant in a room where the temperature is as high as the nurse in charge of it is able to endure. This procedure is necessarily a very uncomfortable one for the nurse, and at times renders it almost impossible for her to use her mind intelligently. It also requires a much more frequent change of nurses than would be the case if the atmosphere of the room were cooler. Another means of preventing undue loss of heat is to wrap the infant in cotton-wool and place it in a basket lined with hot-water bottles.

Incubator.—If possible, the infant should be placed in an apparatus which is called an incubator. These incubators have been used for many years in different parts of the world, notably in Paris. They are of different forms, some better adapted than others for carrying out the different factors of the problem. Some of them are made of tin, with double walls, so that hot water can be continually kept in them, and thus sufficient warmth be applied to the infant. Others are made of wood, and kept warm by means of hot-water bottles introduced into them from below.

The name incubator has been applied to these various devices for keeping up the animal heat of the infant. It is a misnomer, for incubation means hatching, and in the precise sense of the word, the premature infant is already hatched and has been incubated. What we accomplish by this apparatus is analogous to what is done to keep up the animal heat and preserve the lives of young chickens after they are hatched, and the name *brooder* would be more applicable to machines devised for preserving the lives of premature infants than the term *incubator*. The word incubator is, however, so generally used to represent an apparatus intended to preserve the premature infant's life until it has attained the age of two hundred and eighty days, that it will, in all probability, for the present be retained.

When it is impossible to obtain an incubator at once for preserving the premature infant's animal heat, it can be treated in the way already referred to, by placing it in a room in which the temperature has been raised as high as can be comfortably borne by the nurses, and in a basket heated by hot-water bottles. Such a case is represented in Fig. 65.

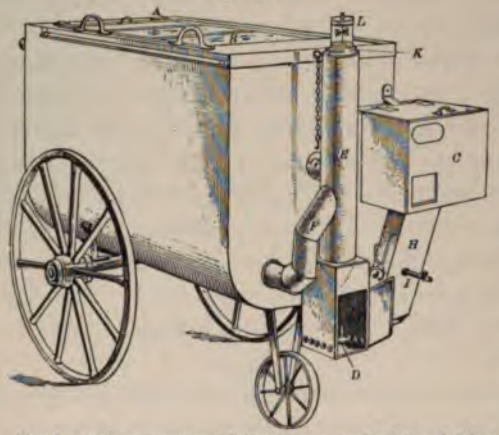
FIG. 65.



Infant premature at twenty-eighth week. Birth-weight, 1200 grammes. Age, 14 weeks. Treated in basket heated by hot-water bottles. Temperature of air in basket shown by thermometer introduced between the side of the basket and the blanket.

Fig. 66 represents an incubator devised by me with the aid and counsel of Mr. G. E. Gordon and Mr. J. P. Putnam. It is intended to cover the requirements needed to preserve the lives of premature infants. It should be made as large as possible. It is, however, so expensive that incubators of wood, though objectionable for many reasons, especially that of infection, will be more commonly used. It is always, however, my wish, if possible, to use what is best, no matter what the cost, where such an important and difficult problem as the saving of a premature infant's life is to be solved. The expense of such an incubator as this one, while too great for any one individual, is comparatively insignificant for a number. The incubator at present must necessarily be an expensive machine, but if provision should be made for it in combination with such scientific facilities for infant feeding as already recommended, I believe that any community would find it of infinite benefit. I am also sure that there would result saving of life for the people, and saving of time and expense for the physicians, combined with the greatest satisfaction to both people and physicians. Such a combination—in cities of a milk-laboratory, or in the country of a Babcock fat-tester, with an incubator, kept in one central station—I hope to see established everywhere. One such station for districts which might be included in a radius of ten or even of twenty miles would be amply sufficient to accomplish very favorable results.

FIG. 66.



Incubator for premature infants. *A*, scales for weighing infant; *B*, glass lid of incubator; *C*, fresh-air box, containing clock-work and fan; *D*, lamp for heating water-jacket; *E*, chimney; *F*, return flue from heating flues; *G*, return fresh-air flue; *H*, entrance for fresh air; *I*, connection for oxygen tank; *J*, mixing valve; *K*, ventilating exit; *L* anemometer.

The incubator (Fig. 66) is intended to fulfil the conditions of a house for the premature infant, and it practically meets the indications called for on page 265. After being used, it can be completely disinfected and cleansed. For purposes of disinfection, and that it may not absorb micro-organisms or dirt of any kind, which in wooden receptacles invariably cause a decided odor, it is made entirely of metal.

It is supported on three wheels, preferably made of light steel, two behind and one guiding wheel in front. A handle is used to push it to different parts of the room, or, if necessary, to an adjoining room, so that the mother can see her infant if she is too sick to leave her bed. The top of the incubator is about 91 cm. (3 feet) from the floor, so that the nurse does not have to stoop unnecessarily, but at the same time can, when sitting down, see into it from above. The body is made of copper; the walls are double, and insulated on the outside, to prevent radiation. The water used for heating circulates on all sides, and the infant is thus warmed by direct radiation. The top of the incubator is covered in the middle by a thick plate-glass lid, which can be raised sufficiently to allow the hands and arms of the nurse to be freely used, and is by a simple contrivance kept from falling down while the infant is being fed or cleansed. A chain prevents the lid from falling backward. On the under side of the glass lid is a fine wire sliding screen, which comes directly over the infant's head and between it and the glass. This is simply a precaution against the possible breakage of the glass lid and consequent injury to the infant.

The plated box (C) attached to the upper front end of the incubator contains some strong clock-work with a fan attachment. The oval opening in the clock-box admits the air to the incubator. Below the opening for the fresh air is a window, through which the fan and clock-work can be watched.

Just below the air-opening and above the clock-work is a fine open wire shelf, on which is spread a thin layer of cotton-wool. The air, which by means of the fan is drawn into the box, is sifted through the cotton and carried down the air-shaft (H) directly into the incubator. In this air-shaft (H) there is a small stop-cock (I). This is the point of attachment for the tube from the oxygen tank, to be used when oxygen is needed to be mixed with the entering air-supply. The admixture of oxygen with the air in the incubator I have found of great value when the infant is losing in weight.

In this air-shaft, also, is attached a valve, which is so regulated by a register handle that the air can be utilized either above or below the boiler, according as it is needed.

The bottom of the incubator constitutes an air-chamber, and in this is a boiler which, with its heating or combustion direct and return flues, warms the interior of the apparatus.

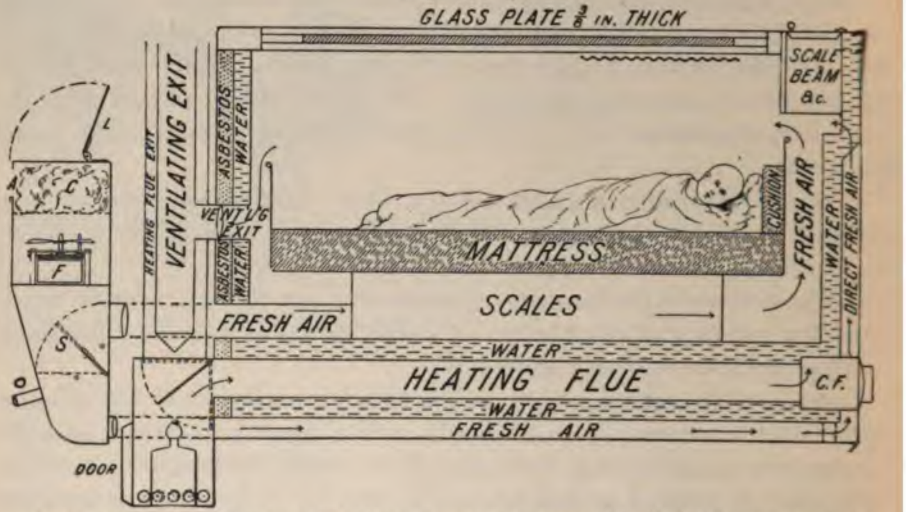
Above the boiler is placed the platform of a scales. To have the infant's bed continuously on a scales is of very great importance, as while seeing from hour to hour the variations in weight the danger of handling the infant to weigh it is obviated, and it need never be removed from the incubator. The balance power of the scales is on the top of the back end of the incubator. The platform of the scales acts as the support for a metal pan on which the infant is placed. This pan should be made of sheet iron, enamelled on both sides with white porcelain enamel, and should have handles at either end to facilitate its removal from the incubator. From the ends of this pan is hung by wires, which can be easily attached or detached, a light frame made of four steel rods crossed. On this frame is tied with tapes a piece of strong cotton cloth. This cloth is the bed, on which the infant is placed wrapped in clean absorbent cotton. This cotton cloth is about 2.5 cm. (1 inch) above the bottom of the pan. The infant's head is turned to the back end of the incubator.

At the front end, opposite the foot of the infant's bed, is the exit (G) for the vitiated air. This exit passes through the end of the incubator and enters a ventilating pipe which has at its top an anemometer (L). The bottom of the shaft is outside the incubator, and has a closed cone-shaped end, which is enclosed in a metal box in such a way that a lamp (D) can be placed under it. The heat from this lamp answers two purposes; one of which is, by keeping the ventilating shaft hot, to aid the ventilation, and the other is to heat the water in the boiler. A register-valve (J) attached to the pipe can shut off the heat if necessary from the boiler, and allow it to go directly up the double pipe (*E*, *K*), whereby its entire power will be used in promoting ventilation, or the valve may be set so as to direct the flame partially into the boiler, thus placing its temperature completely under control. In this way the heat from the lamp (which is enclosed in the box) is without danger entirely utilized for heating and ventilation.

Fig. 67 shows a section of the incubator.

The smoke-flue of the lamp, marked "Heating Flue," passes through the centre of the boiler, marked "Water" in the diagram, as far as to the cleaning-flue, marked *C. F.* Thence it returns and enters the upright pipe, marked "Heating Flue Exit." The horizontal return-flue is not shown in the figure, because it is behind the horizontal arm. The little gate-valve shown directly above the lamp regulates absolutely the amount of heat which is allowed to pass through the boiler, and the temperature of the warm water therein may be tested by a thermometer, inserted at any opening which may be provided for it as directed when the incubator is built.

FIG. 67.



Section of incubator. *L*, lid of fresh-air box, open; *A*, entrance of fresh air; *C*, cotton, resting on wire shelf above clock-work; *F*, clock-work and fan; *S*, valve regulating hot and cold fresh air; *O*, pipe for oxygen attachment; *C. F.*, cleaning flue; *Door*, door to lamp-box; *W*, wire frame to protect against breakage of lid.

The fresh-air flues are constructed as shown, one above and one below the boiler. One flue comes in contact with the upper or hottest part of the boiler, and presents a very large surface of contact by being flattened so as to cover completely the upper side of the boiler. The other flue touches the bottom of the boiler only in one line, or not at all, so that the air passing through it is practically unaffected by the boiler heat. By this arrangement the temperature of the fresh air can be regulated at will by the attendant by simply raising or lowering the valve *S*.

In virtue of the large amount of heating surface of the heating flue in this apparatus, it is found that a very small flame suffices to keep up the desired temperature, and it results from this that no injurious products of combustion contaminate the air of the room. A very small alcohol lamp can be used, while with a less scientific arrangement this fuel might be found too expensive.

It is probable that an electric current will be found most suitable to supply the heat in place of the lamp, as well as to drive the fan, and this can be very easily accomplished with a small battery.

By packing the water-jacket with asbestos, external radiation is prevented.

The heating of the incubator varies as to time and degree according to the atmosphere of the room where it has been standing. If, however, the temperature of the room is 21.1° C. (70° F.), and the temperature of the water which is introduced into the boiler is about 40.5° C. (105° F.), it will be found that after the cool air in the incubator has been displaced the temperature of the air in the incubator will in about fifteen minutes rise to 35° C. (95° F.). The temperature will remain at this point for about half an hour. As soon as the temperature begins to fall the alcohol lamp should be lighted, and as soon as the temperature of the water in the boiler rises above 35° C. (95° F.) the lamp should be extinguished. By careful regulation of the lamp and of the fresh air by means of the register-valves, an intelligent nurse can keep the temperature of the incubator at whatever degree the physician orders. The thermometer should, in order to show accurately the temperature of the air which the infant is breathing, be beside it on its bed, as when attached to the lid it is influenced by changes of temperature in the room.

If any difficulty arises from the temperature not responding quickly enough to the register-valves and lamp, it is well to draw off a little hot water and replace it by some cold water if it is desired to lower the temperature, while to raise the temperature the withdrawn water is to be replaced by hot water. In practice it has been found that this latter method of regulating the temperature is more satisfactory than by the lamp. If the incubator is not kept in one place, as a hospital, but has to be transported, it would be well to lighten it as much as possible by removing the wheels and making it of some metal lighter than copper, as it is very heavy.

Nurses.—The incubator is not intended to obviate the necessity of skilled nursing. On the contrary, a nurse should be in constant attendance night and day. She should have all the details of the infant's care and the mechanism of the incubator explained to her minutely, for an emergency may arise at any time, and always requires to be dealt with immediately.

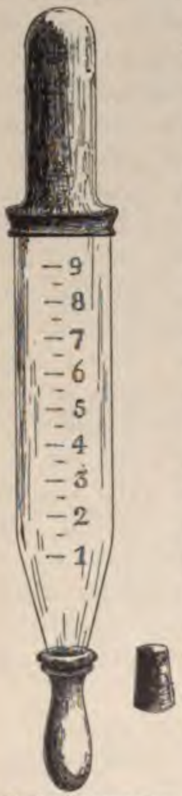
The incubator supplies the means for exact treatment, but intelligent minds and trained gentle hands are indispensable. The nurse should frequently observe the infant through the glass lid, and should be certain that the anemometer is in constant motion.

Apparatus Connected with the Incubator.—A stethoscope with rubber arms and small cup is the best adapted for examining the infant in the incubator, as it can be bent in any direction, and is suited to the infant's size.

A piece of dark cloth should be kept over the glass lid, to exclude the light, while the sun should be allowed to shine freely into the room. The air in the room should be frequently changed and kept as pure as possible. The room should be absolutely free from dust and should only contain some polished wood chairs and tables. The floor should be bare, and, if possible, of polished hard wood.

The method of feeding the infant in the incubator is important. It frequently happens that the premature infant is too weak not only to suck the breast, but also to be fed from the bottle. In such cases it is customary to use a spoon or a medicine-dropper. These, however, are very unsatisfactory instruments. The food is liable to be spilled, the spoon or dropper has to be frequently filled, and much time is taken to complete the feeding. The lid of the incubator, also, should not be kept open for a longer time than is unavoidable. I have lately made use of a device suggested by Dr. Breck. It is simply a glass cylinder (Fig. 68), 12 cm. (4¾ inches) long and 2.4 cm. (1 inch) in diameter. The cylinder is graduated to 2 c.c. (½ drachm), and holds 36 c.c. (9 drachms). It is shaped at one end so as to have a small rubber nipple fitted to it. The large end is covered by a rubber cot. The rubber cot, which has no holes, acts as an air-reservoir, and by simply introducing the small perforated nipple into the mouth and gently pressing the rubber cot the food is slowly forced down the infant's throat, without choking it and without obliging it to suck or apparently to use any effort. To fill the tube the rubber nipple and cot are removed, and the required amount of food is poured in at the large end, while the small end of the cylinder is plugged with a rubber stopper shown in the figure beside the feeder.

Fig. 68.



Feeder for premature infants (reduced one-half).

This method of feeding is especially desirable for a weak premature infant in an incubator, because it entails no loss of strength on the part of the infant, and can be easily managed by the right hand of the nurse, while her left hand supports the infant's head. This method is far preferable to that of gavage, which is not so easily managed by the nurse and is more exhausting to the infant.

Food.—The feeding of premature infants should be as exact as possible. If the infant is vigorous enough to suck, and if the breast-milk is of proper quality, that is not too strong, the infant can be put to the breast every one and one-half hours. We must consider, however, certain disadvantages of nursing premature infants which do not arise in the nursing of infants at term. Frequent nursing, such as every hour, which is so often required with premature infants, tends to disturb the quality of the breast-milk and to increase the solids. This is often disastrous to the infant, as the least overtaxing of its digestion may prove fatal. Then, again, the premature infant can often digest only much lower percentages of fat, sugar, and proteids than are found in normal breast-milk, so that a normal milk may prove fatal by being too strong. If two or even three wet-nurses can be obtained, the intervals of nursing each one of them can be made every three hours, and thus while the solids in their milk will be lessened, the infant can be fed every hour. This method of feeding if possible is desirable. The laboratory method of feeding with exact and low percentages gradually increased is the most rational and practical, especially if the infant is too weak to nurse at the breast.

Amount.—The amount of food to be given at each feeding is very important. By referring to the weights and gastric capacities of the premature infants already described (Figs. 60, 61, 62, page 260) it will be seen how misleading is the weight of the infant if we take it as an exact index of the gastric capacity. We must, however, take the weight into account, as, from even the imperfect data at our command, the weight of premature infants appears to bear a decided relation to their gastric capacity. We should at least be more inclined to increase rapidly the initial amount of food given in the case of an infant of large weight than in that of a small one. It is better to begin with too small rather than too large a quantity. By watching carefully for signs of hunger, a desire which the infant expresses by feeble but continuous cries, which stop when the food is given to it, we can gradually increase the amount until it seems to want it at regular intervals, is satisfied, and sleeps quietly during the intervals of feeding.

By referring to the fœtal stomachs represented in Figs. 60, 61, 62, 63, pages 260, 261, it will be understood that it is safer to begin with 4 or 5 c.c. (about 1 drachm) and gradually to increase the amount up to a point where our very imperfect knowledge on this subject, derived partly from the weight of the infant, makes us believe that the stomach is full, than to begin at once with the larger amount. It is absolutely necessary that we should avoid undue distention of the stomach, as this may prove fatal.

Intervals.—The premature infant's stomach is small, and is, in all probability, emptied quickly, and, as food is necessary for keeping up the animal heat required for the maintenance of its life, the intervals of feeding should be much shorter than those required for the infant at term. In the early days, and in fact weeks, of life I have found that it is better to feed the premature infant regularly every hour. Four or five week birth, if it is gaining in weight and is digesting well, these intervals lengthened, and by the time it arrives at term we can usually ma feeding intervals one and a quarter to one and a half hours, and weeks later two hours.

Percentages.—The careful adjustment of the premature infant' to its digestive organs is of even greater importance than in the case infant at term. There is no doubt that if we consider the hypersen condition and the undeveloped state of the digestive organs prior to the most exact adjustment of the food to these digestive organs is lutely necessary. This adjustment is best accomplished by means of fully prepared prescriptions at the milk-laboratory. Through this in ment of precision three important advantages are gained: (1) we i a clean food free from micro-organisms; (2) we can obtain low and erly balanced percentages of the constituents of the milk; (3) we hav any time, the power of exactly varying, to within a fraction of one-ha one per cent., the percentages of the three most important element the milk,—namely, the fat, the sugar, and the proteids. In additio these latter two advantages possessed by the substitute over the mate method of feeding are others of almost equal importance. One advan is the absence of variation in the substitute food arising from emotio causes, and another is that the infant need not be taken from the incub to be fed.

The following prescription is the one which I should begin with in fe ing an infant premature at the twenty-eighth week:

PRESCRIPTION 42.

℞ Fat	1.00
Sugar	3.00
Proteids: lactalbumin, 0.25; caseinogen, 0.25	0.50

24 meals, each 4 c.c. (1 drachm).
Heat to 68.3° C. (155° F.).
Reaction faintly alkaline.

If the infant is over twenty-nine weeks, or if it is unusually large its age, and especially if it is unsatisfied, it is well in a few days to cha the prescription to the following:

PRESCRIPTION 43.

℞ Fat	1.50
Sugar	4.00
Proteids: lactalbumin, 0.25; caseinogen, 0.25	0.50

24 meals, each 8 c.c. (2 drachms).

If the infant is over thirty-two weeks, the prescription should changed in a few days, under the same conditions as in Prescription to

PRESCRIPTION 44.

℞ Fat	1.50
Sugar	5.00
Proteids: lactalbumin, 0.50; caseinogen, 0.25	0.75

24 meals, each 12 c.c. (3 drachms).

If the infant is over thirty-six weeks, the milk should, after forty-eight hours, be changed to

PRESCRIPTION 45.

℞ Fat	2.00
Sugar	5.50
Proteids: lactalbumin, 0.75; caseinogen, 0.25	1.00

24 meals, each 16 c.c. (4 drachms).

The infant, however, under all circumstances, must be watched critically, and any or all of the percentages of the elements or amounts of the food increased or decreased according to the individual indications.

When the infant is born at the thirty-eighth or thirty-ninth week its development is usually so near that of the infant at term that the incubator will not be needed, and the food can be given in about the proportions which would be adapted to the early days of the infant at term (Prescription 16, page 215).

PROGNOSIS.—The prognosis in a premature infant during the early weeks of its life and until it is steadily gaining in weight must be guarded, as the mortality rate is very high, and these infants are apt to die suddenly simply from a lack of vitality. The younger the infant the greater is the danger of its dying. The most frequent causes of death among premature infants are a lack of development of the thorax or lungs, atelectasis, an insufficient supply of fresh air, improperly cleansed incubators, leading to various forms of infection, undue exposure, careless handling, and improper feeding. Hutinel, from his experience with premature infants at the Hospice des Enfants-Assistés, attributes the high mortality to the following causes:

(1) Some infants are not viable; their organs being incompletely formed, cannot perform the functions necessary for the maintenance of life.

(2) Others present malformations inconsistent with life, or are affected with an hereditary taint, such as syphilis, which has already done irreparable damage at the time of birth. In these two cases the incubator is useless; if it protects against cold, which is all it can do, it cannot perfect undeveloped organs or cure hereditary blemishes.

(3) Some babies which seem well at birth fall ill soon after they are put into an incubator, and die there. In these cases the incubator is not to blame.

(4) This class includes premature infants who are apparently well developed and comparatively healthy when placed in the incubator. It seems as if they ought to develop, but they die after a few days. The

author considers that they die from various infections. Thes simple surface infections, for the actual presence of the pus- organisms in the blood have been frequently demonstrated. T tions are due to the lack of resisting power on the part of the mucous membrane which cannot oppose the entrance of gen surrounding atmosphere is contaminated. That the air comes side is not sufficient to eliminate contagion, as the incubator opened from time to time, and the patients are attended by t may readily infect them. The author insists most strongly upo ventilated and sunny room for incubators, and holds that in there should be at least three apartments devoted to the rearin mature infants: one for the healthy, one for the ailing, and on ill ones. Germs undoubtedly flourish in an incubator with its c elevated temperature, so that an infant should be removed just as it is able to maintain its temperature at 98.4° F. for thirty-six eight hours. It should then be enveloped in cotton and surrou hot bottles. Plenty of fresh air and sunshine are required. If on babies falls ill it must be separated at once from the others. In putting it back into the incubator, infection when it occurs is m to begin outside. Incubators should be so constructed that the readily cleansed. The temperature changes involved in doing tl little importance provided they are not too prolonged. By follow principles the author has lost only three out of twenty-one ca aging less than four and a half pounds.

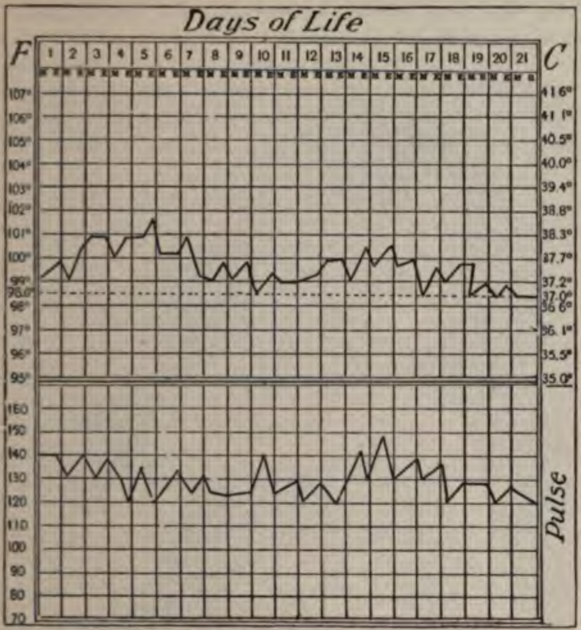
The following record and chart are those of a premature ir was kept in a room with a temperature of 23.8° C. (75° F.) basket heated to 29.4° C. (85° F.).

TABLE 59.

Weight for Sixty-one Days of Infant Premature at Thirty-two Weeks.

Day of Life.	Grammes.	(Pounds.	Oz.)	Remarks.
Birth-weight	2964	(6	8)	Cow's milk, with spoon.
Third	2724	(6	0)	Mother's milk, with spoon
Sixth	2814	(6	3)	Mother's milk, with spoon
Ninth	2964	(6	8)	Mother's milk direct from
Thirteenth	3178	(7	0)	Mother's milk direct from
Sixteenth	3388	(7	7)	Modified milk.
Twentieth	3598	(7	14)	Modified milk.
Twenty-third	3812	(8	6)	Modified milk.
Twenty-seventh.....	4116	(9	1)	Modified milk.
Thirtieth	4236	(9	5)	Modified milk.
Thirty-third..	4476	(9	13)	Modified milk.
Thirty-seventh......	4600	(10	2)	Modified milk.
Forty-first..........	4840	(10	10)	Modified milk.
Forty-fourth........	4900	(10	12)	Modified milk.
Forty-eighth	4994	(11	0)	Modified milk.
Fifty-first..........	5084	(11	3)	Modified milk.
Fifty-fifth..........	5234	(11	8)	Modified milk.
Fifty-eighth........	5324	(11	11)	Modified milk.
Sixty-first..........	5384	(11	13)	Modified milk.

CHART 3.



The question is often asked whether premature infants, even if their lives are saved, can eventually be as well developed physically and mentally as are those born at term. In my experience there seems to be no question that when once we have succeeded in making the infant gain steadily in weight and assume the appearance of an infant at term its subsequent condition differs in no respect from that of infants born at term. Fig. 69 illustrates the wisdom of doing all in our power to save the lives of these infants, as it represents at nine months what was one of the worst cases of lowered vitality and extreme emaciation in a premature infant that I have ever seen.

This picture was taken when the infant was nine months old and weighed 8400 grammes (17½ pounds). As its birth-weight was 2040 grammes (about 4½ pounds), it will be seen that its weight has quadrupled. He was fed entirely on modified milk from the laboratory during the first year, and was a fine large boy, walking and talking at two years of age. He was perfectly healthy and well developed both physically and mentally.

His sister, who was premature at the twenty-eighth week, was eight years old. She was well developed and strong, and was unusually bright and intelligent for her age. She was, in fact, decidedly in advance mentally of the other children of her age at school.

The following is the record of an infant prematurely born at about the thirtieth week, and weighing 2850 grammes (about 5 pounds 15 ounces), which was the first premature infant that happened to be treated in the incubator represented in Fig. 66, page 268. It illustrates the value of careful and exact incubation and feeding.

The infant was born at ten minutes past three in the morning of February 16. It was placed in the incubator at 9 P.M. of the same day, the temperature of the incubator being 34.4° C. (94° F.).

FIG. 69.

Infant premature at 30 weeks. Birth-weight, 2040 grammes (4¼ pounds.) Treated in incubator 64 days. Age, 9 months; weight, 8400 grammes (17½ pounds.)

On the following day, February 17, the infant was given by the nurse 4 c.c. (1 drachm) of diluted cow's milk every hour for three feedings, which he vomited almost immediately after taking. The intervals of feeding were then increased to two hours, but the milk was not retained. The nurse then gave him 2 c.c. (½ drachm) every three hours during the night, which he retained for a number of feedings, but then vomited bile and mucus, together with the undigested food which had been given him.

February 18 the infant was found to have lost 420 grammes (14 ounces) in weight, to be very weak, and to be unable to retain the milk diluted with water. The meconium came away on this day, and there was a uric acid stain on the napkins. The infant was very restless. Its respirations were irregular, and its feet and hands were cold. The temperature of the incubator, which up to this time had been kept at 34.4° C. (94° F.), was lowered to 33.8° C. (93° F.), as the infant had begun to perspire. A substitute food was ordered from the milk-laboratory on this day, the prescription for which was as follows:

PRESCRIPTION 46.

℞	Fat	1.00
	Sugar	3.00
	Proteids	0.50
	To be heated for thirty minutes at 75° C. (167° F.).	
	Lime-water	5.00
	24 tubes, each containing 4 c.c (1 drachm).	

This food was given to the infant every hour.

On the following day, the 19th, the record was that the food had been retained, that the infant had seemed so hungry that the amount had to be increased to 10 c.c. (2½ drachms), and that it was found advisable to feed it every two hours rather than every hour. There was no vomiting. There were two movements of the bowels, which still showed evidences of undigested milk and some meconium. The infant's weight on this day was found to be the same as on the previous day, 2300 grammes (5 pounds 1 ounce).

On the following day, February 20, the infant was found to have gained 30 grammes (1 ounce). It was taking its food regularly every two hours, alternating with the mother's milk, which had come in considerable quantity. There were still evidences of uric acid in the urine. The temperature of the incubator was kept at 31.6° C. (89° F.).

On the following day, February 21, the weight was as on the previous day, 2230 grammes (5 pounds 2 ounces). The color of the fæcal discharges was yellowish brown. There was only one discharge in the twenty-four hours, obtained by the use of a suppository. The temperature of the incubator was kept at 30° C. (86° F.).

On the following day, February 22, it was found that the infant had lost 60 grammes (2 ounces). The substitute food was then given every two hours, alternating with the breast-milk. On that day there were three yellow well-digested movements. The temperature of the incubator was kept at 29.4° C. (85° F.). The infant seemed stronger, was very quiet, and slept except when it awoke to receive its food.

On the following day, February 23, there is no record of the infant's weight, but it was evidently in a very precarious condition and seemed exhausted. It did not take its nourishment readily. It had five small fæcal discharges in the twenty-four hours, which, however, were yellow and fairly digested.

On the following day, February 24, the breast-milk was omitted, and 4 c.c. (1 drachm) of modified milk were given every two hours, the percentage of the sugar being raised from 3 to 3.5. There were four small fæcal movements during the day; the first one was green, the last three were yellow and decidedly better digested. The temperature of the incubator was kept at 29.4° C. (85° F.). During the day the infant gained 60 grammes (2 ounces) in weight. It was so weak on these two days that it would have been dangerous to take it out of the incubator to weigh it, so that the continual record of the weight which could be obtained by the scale-bed of the incubator was of the utmost value in regulating the changes in the food necessary to save the infant's life.

On the following day, February 25, the infant's weight was found to be 2260 grammes (5 pounds 3 ounces), an increase of 30 grammes (1 ounce). The percentages in the modified milk were then changed to the following:

PRESCRIPTION 47.

℞	Fat	1.50
	Sugar	4.00
	Proteids	0.75

One drop of brandy was given with each feeding. There was one fæcal discharge, which was yellow and well digested. On this day 4 c.c. (1 drachm) of food were given to the infant every two hours until its feeding at 10.30 P.M. After this it seemed so hungry that at midnight 36 c.c. (9 drachms) were given, at 3 A.M. 40 c.c. (10 drachms) were given, and at 5.30 A.M. 30 grammes (1 ounce) were given. The weight was now found to be 2420 grammes (5 pounds 5 ounces), an increase of 60 grammes (2 ounces) in the twenty-four hours. The amount of food which the infant had taken in the previous twenty-four hours was found to have been 375 grammes (12½ ounces). The fæcal discharges were yellow and well digested. Brandy was continued.

The temperature of the incubator was kept at 29.4° C. (85° F.). At times a breast-milk was given to the infant, in order to satisfy the mother, but it evi did not agree with it.

On February 27 the weight was found to be 2450 grammes (5 pounds 6 ou The prescription for the modified milk was then changed as follows :

PRESCRIPTION 48.

℞ Fat	2.00
Sugar	5.00
Proteids	0.75

Thirty grammes (1 ounce) of this were given to the infant every two hours du the day, and every two and one-half hours during the night. One yellow well-dige fæcal discharge was obtained by means of a suppository. The temperature of th cubator was then reduced to 27.7° C. (82° F.).

The following day, February 28, the weight was found to be 2480 gramme pounds 7 ounces). The brandy was still continued, and there was one yellow w digested fæcal discharge. The breast-milk had been entirely omitted, and 450 gram (15 ounces) of modified milk had been taken in the twenty-four hours.

On the following day, March 1, it weighed 2510 grammes (5 pounds 8 ounc The amount of modified milk given was 495 grammes (16½ ounces) in the twenty-f hours, and one drop of brandy was given with each feeding. There was great provement in the infant's appearance, and it was much stronger.

On the following day, March 2, there had been no increase or loss in weight. T temperature of the incubator was kept at 27.2° C. (81° F.). 510 grammes (17 ounc of the modified milk were taken in the twenty-four hours. There was one fæ movement, well digested and yellow.

On the following day, March 3, the weight was found to have increased to 26 grammes (5 pounds 11 ounces). The percentages of the modified milk were th changed to the following :

PRESCRIPTION 49.

℞ Fat	2.50
Sugar	5.00
Proteids	1.00

There were two well-digested fæcal discharges on this day. The temperature o the incubator was reduced to 25° C. (77° F.). 615 grammes (20½ ounces) of the modified milk were given in the twenty-four hours.

The following day, March 4, the infant was found to have lost 60 grammes (ounces), and the temperature of the incubator was therefore raised to 26.6° C. (80° F.). 630 grammes (21 ounces) of modified milk were taken in the twenty-four hours, and there was no especial change in the infant's condition.

On the following day, March 5, 30 grammes (1 ounce) in weight were found to have been gained, and the infant was looking better and decidedly gaining in strength. It was evident that the proper temperature for this especial infant at this age and at this period of its development was 26.6° C. (80° F.).

After this time the infant continued to develop normally, and on being taken out of the incubator in April was thriving in every way.

When five months old it weighed 7110 grammes (14 pounds and 13 ounces).

The next case was that of an infant which was four weeks premature, and which was, for a premature infant, tolerably vigorous at birth. It was under the care of Dr. Samuel Breck, with whom I saw it in consultation. It was not placed in an incubator. Unfortunately, its nurse had no idea of the importance of protecting it from external influences. It was fed on a carefully prepared food from the milk-laboratory, and began to gain in weight, and in every way showed no evidence of its vitality being interfered

Showing Detail

DAYS OF LIFE.	INTERVALS BETWEEN MEALS.	AMOUNT AT EACH MEAL.		PERCENTAGES OF FOOD.				FÆCAL		
		C.c.	Dr'ms.	Fat.	Sugar.	Proteids.	Lime-Water.	No.	Char	
1	. . . .	. . . .	. . . . .	. .	3 per ct. sol. in aq. dis.	. . . . .	. . . . .	. .	meconi	l. Lungs normal. Em
2	1 hour.	4	1	1.00	3.00	1.00	5.00	2	brown,	C. (167° F.).
3	1 hour.	4	1	1.00	3.00	1.00	5.00	2		
4	1 hour.	4	1	1.00	3.00	1.00	5.00	2		
5	1 hour.	4	1	1.00	3.00	1.00	5.00	2	only digest	pired freely. Temper iccough relieved by br
6	1 hour.	4	1	1.00	3.00	1.00	5.00	2		n imperceptible for 10
7	1 hour.	4	1	1.00	3.00	1.00	5.00	2		ittle, temperature of in
8	1 hour.	4	1	1.00	3.00	1.00	5.00	2		neonatorum. Black c
9	1 hour.	4	1	1.00	4.00	1.00	5.00	2	yellow,	
10	1 hour.	4	1	1.00	4.00	1.00	5.00	2	well di	
11	1 hour.	8	2	1.00	4.00	1.00	5.00	2		
12	1 hour.	8	2	1.00	4.00	1.00	5.00	2		
13	1 hour.	8	2	1.00	4.00	1.00	5.00	2		sh-air box for 10 minut
14	1 hour.	8-10	2-2½	1.00	4.00	1.00	5.00	2		Oxygen as on 13th.
15	1 hour.	8-10	2-2½	1.00	4.00	1.00	5.00	2		
16	1 hour.	12	3	1.50	5.00	1.00	5.00	2		when incubator below
17	1 hour.	12	3	1.50	5.00	1.00	5.00	2		
18	1 hour.	12	3	1.50	5.00	1.00	5.00	2		
19	1 hour.	12	3	1.50	5.00	1.00	5.00	2		
20	1 hour.	12-14	3-3½	1.50	5.00	1.00	5.00	2		
21	1 hour.	16	4	1.50	5.00	1.00	5.00	2		
22	1 hour.	16	4	1.50	5.00	1.00	5.00	2		
23	1 hour.	16-18	4-4½	1.50	5.00	1.00	5.00	2		
24	1 hour.	16-18	4-4½	1.50	5.00	1.00	5.00	2		
25	1 hour.	18-20	4½-5	2.00	5.00	1.00	5.00	1		
26	1 hour.	18-20	4½-5	2.00	5.00	1.00	5.00	2		
27	1 hour.	18-20	4½-5	2.00	5.00	1.00	5.00	1		
28	1 hour.	18-20	4½-5	2.00	5.00	1.00	5.00	2		
29	1 hour.	20	5	2.00	5.00	1.00	5.00	2		
30	1 hour.	22	5½	2.00	5.00	1.00	5.00	3	slightly	
31	1 hour.	22	5½	2.00	5.00	1.00	5.00	4		
32	1 hour.	22	5½	2.00	5.00	1.00	5.00	4		
33	1 hour.	24-26	6-6½	2.00	5.00	1.00	5.00	5		emperature of incubator
34	1 hour.	28	7	2.00	6.00	1.00	10.00	8		ly.
35	1¼ hrs.	32	8	2.00	6.00	1.00	10.00	5	yellow well di	en. Began to feed with
36	1¼ hrs.	32	8	2.00	6.00	1.00	5.00	5		Slight cyanosis. Oxyge
37	1¼ hrs.	32	8	2.00	6.00	1.00	5.00	3		
38	1¼ hrs.	48	12	2.00	6.00	1.00	5.00	3		
39	1¼ hrs.	48	12	2.00	6.00	1.00	5.00	3		
40	1¼ hrs.	48	12	2.00	6.00	1.00	5.00	5		ding.
41	1½ hrs.	48	12	2.00	6.00	1.00	5.00	2		
42	1½ hrs.	48	12	2.00	6.00	1.00	5.00	2		
43	1½ hrs.	48	12	2.00	6.00	1.00	5.00	1		or. Omit oxygen.
44	1½ hrs.	48	12	2.00	6.00	1.00	5.00	2		
45	1½ hrs.	48	12	2.00	6.00	1.00	5.00	5		
46	1½ hrs.	48	12	2.00	6.00	1.00	5.00	6		
47	1½ hrs.	48	12	2.00	6.00	1.00	5.00	6		
48	1½ hrs.	48	12	2.00	6.00	1.00	5.00	4		
49	1½ hrs.	48	12	2.00	6.00	1.00	5.00	4		
50	1½ hrs.	56	14	2.00	6.00	1.00	5.00	6		
51	1½ hrs.	56	14	2.00	6.00	1.00	5.00	5		
52	1¾ hrs.	64	16	2.00	6.00	1.00	5.00	6		
53	1¾ hrs.	64	16	2.00	6.00	1.00	5.00	5		
54	1¾ hrs.	64	16	2.00	6.00	1.00	5.00	2		
55	1¾ hrs.	64	16	3.00	7.00	1.00	5.00	1		
56	1¾ hrs.	64	16	3.00	7.00	1.00	5.00	1		er and brighter, and is tra
57	1¾ hrs.	64	16	3.00	7.00	1.00	5.00	3		
58	1¾ hrs.	64	16	3.00	7.00	1.00	5.00	1		
59	1¾ hrs.	64	16	3.00	7.00	1.00	5.00	3		
60	1¾ hrs.	64	16	3.00	7.00	1.00	5.00	1		
61	1¾ hrs.	64	16	3.00	7·00	1.00	5.00	3		mited. Put back into incu
62	1¾ hrs.	64	16	3.00	7.00	1.00	5.00	5		
63	1¾-2 hrs.	64	16	3.00	7.00	1.00	5.00	2		
64	1¾-2 hrs.	64	10	3.00	7.00	1.00	5.00	2		ater at 35° C. (95° F.).
65	1¾-2 hrs.	64	16	3.00	7.00	1.00	5.00	2		tronger.
68	1¾-2 hrs.	64	16	4.00	7.00	1.00	5.00	2		

At six months weighed 7000 grammes (14 pounds lbs.

with ; but the nurse was possessed with the idea that it needed plenty of cold fresh air. The window in the infant's room was left open one night when the weather was quite ool. The following day it did not take its food well, was somewhat cyanotic, and was ound to have lost almost 240 grammes (½ pound). It was then placed, as it should have een in the beginning, in a warm room, treated with the utmost care, and not handled uch. None of these measures, however, were sufficient to prevent a still further les-ening of its vitality. It never rallied from the first blow which was struck at its tality, and lost its life practically through the ignorance of the nurse who was in arge of it.

A post-mortem examination showed nothing abnormal, except that the mesenteric ands were somewhat enlarged.

The next case was that of an infant born at about the twenty-fifth week of intra-terine life. Its weight was 1080 grammes (about 2¼ pounds). There were a number f interesting points to be recorded in this case.

It was not strong enough to suck, and had to be fed with a spoon. Its mother's ilk, the analysis of which is given below, at once caused such disturbance that modi-ed milk from the laboratory had to be substituted.

(*Mother's Milk.*)

Fat	1.29
Sugar	4.10
Proteids	6.83
Mineral matter	0.26
Total solids	12.28
Water	87.72
	100.00

The prescription for the modified milk which it digested well was

PRESCRIPTION 50.

Modified Milk.

℞ Fat	1.00
Sugar	3.00
Proteids	0.75

The infant's temperature in the rectum was 36.7° C. (98° F.). It seemed to be doing fairly well, but did not gain in weight, and on the fifth day of its life was unable to swallow. It was then fed by gavage.

It was treated with great care so far as keeping it warm was concerned, but an incubator could not be obtained for it, and it died when it was seven days old.

It is interesting in this case to notice that the meconium came as is usual in the infant at term, and began to change its color on the third day, and that by the fifth day the fæcal movements were yellow and well digested.

Table 60 records the details of an infant's life in an incubator during a period of sixty-four days. This record will be of great use to any one who has charge of a premature infant in an incubator, as it illustrates exactly what emergencies are likely to arise and how they can be met.

The infant, as is seen by referring to the column of remarks, came very near dying a number of times, and unquestionably would have died had it not been carefully managed, as, for example, by the administration of oxygen, by prompt changes in its food, by the regulation of the temperature of the incubator, and by the constant attention of a day nurse and a night nurse.

DIVISION V.

DISEASES OF THE NEW-BORN.

Before entering into a description of the individual diseases of new-born, it will be profitable first to consider some of the general aspe of disease as it occurs in early life.

Disease in General.—The peculiarities which characterize disease children are mostly limited to the period of infancy and the early a middle periods of childhood. In later childhood, that is, after the eigh or ninth year, disease, both in its etiology, pathology, symptoms, and pro nosis, resembles closely that which is met with in adult life.

In the earlier periods of life, on the contrary, we meet with distin differences, depending partly on the great rôle which congenital disease play in the various stages of development and partly on the greater vulnerability of the growing tissues and their lessened power of resistanc not only to the known specific infections but to numberless as yet undifferentiated varieties of pathogenic micro-organisms, which are as yet bu little understood.

Inheritance.—A very important element to be taken into consideration as influencing the tendency to disease is inheritance. The occurrence in the parents of such diseases as tuberculosis, rheumatism, and the various neuroses seems to render the tissues of the children not only more receptive to these conditions but to so vitiate them that they are readily affected by many other diseases. The direct inheritance of such diseases as syphilis and the great mortality arising from them are distinctly characteristic of disease in infancy.

Malformation.—The greater number of diseases and the greater tendency to disease in early life, as compared with a later period, is caused in large measure by a lack of normal intra-uterine development, resulting in malformations, each representing a disease in itself and each having such influence on the tissues in general that they become abnormally receptive to many diseases. These malformations, such as of the mouth, nose, bladder, and rectum, are of especial significance from a surgical point of view, while those which are of especial interest to us medically are malformations of the heart and brain and such conditions of arrested development as are represented by atelectasis.

Traumatism.—A class of diseases distinctly infantile is represented by traumatic causes, such as certain forms of meningeal hemorrhages, abrasions occurring during delivery, which afford, as in the case of an unhealed umbilicus, a ready entrance for the micro-organisms of tetanus and of erysipelas. The various forms of ophthalmia neonatorum must be included in this class of affections.

GENERAL ETIOLOGY.—In addition to the congenital causes of infantile disease just enumerated the etiology of the acquired diseases of early life is of great interest and importance, for there is no doubt that most of the diseases of early life are the direct result of the ignorance or neglect of those who have the charge and direction of infants during this period. The chief etiological factors of disease in infancy are improper food and unhygienic surroundings. These conditions may cause such specific diseases of nutrition as rhachitis, scorbutus, and infantile atrophy, or may result in such a marked degree of malnutrition that there is a decided predisposition to various acute diseases such as those of the gastro-enteric tract and of the lung, and, later, to such neuroses as chorea and many functional disturbances.

GENERAL PATHOLOGY.—The pathological processes which occur in early life, as distinctive from a later period, are essentially acute, the chronic morbid processes, except those which result from acute disease, being rare. Hyperplasia of the lymph-nodes and their susceptibility to infection of all kinds, together with their great activity in carrying infection, are a characteristic feature of infantile pathology.

A series of 726 consecutive autopsies made at the New York Infant Asylum and tabulated by Holt, shows the relative pathology of the different organs. None of these autopsies were under one month, 72 per cent. were under one year, and only 3 per cent. were over two years.

According to these figures the lungs first and the intestine second were found affected in the greatest number of cases, and it was noted that it was rare to find the lungs normal after any acute infectious disease had lasted a week. Out of the above-mentioned 726 cases, pathological conditions were found in the *lung* in 399 cases ; of these, 322 were cases of pneumonia, of which 139 were primary and 56 were tuberculous. There were 6 cases of congenital atelectasis, 1 case of serous pleurisy, 5 cases of empyema, and although there was dry pleurisy in nearly all the severe cases of pneumonia, there was no case of pleurisy uncomplicated by disease of the lung. The *gastro-enteric tract* was affected in 189 cases ; of these, 116 cases were acute ileo-colitis with or without gastritis. There were no cases of gastritis without intestinal lesions. There were 72 cases of acute diarrhœa without gross lesions. There was one case of intussusception. The *brain* and *meninges* were affected in 35 cases, of which 11 were tubercular meningitis. There were 26 cases of malnutrition without gross lesions. The *kidneys* were affected in 26 cases, 7 of which were malformations, and only 5 cases were primary. The *heart* was affected in 6 cases ; of these, 3 were congenital malformations, 3 were pericarditis, all occurring in cases of pneumonia, and there were no cases of acute or chronic endocarditis. The *peritoneum* was affected in four cases. The *mouth* was affected in one case (noma).

It thus appears that pathological conditions of the liver and spleen are rare in early life, as are primary disease of the kidneys and organic disease of the brain itself. New growths are rare, and, when present, are usually of the kidney or bones. Diseases of the bones and joints are very common, and are usually tubercular or syphilitic, more commonly the former. Diseases of nutrition are extremely common.

General Symptoms.—The clinical picture of disease in older children does not present many features different from what is seen in the adult. In infancy and early childhood, however, there are many differences. The infant cannot express its thoughts and sensations in words, and in place of this indicates its discomfort by cries and various movements. In addition to this, the nervous system is in so sensitive a condition and in such unstable equilibrium that its manifestations in disease are very misleading. In disease of the lung, for instance, the young child is very apt to locate its discomfort in the abdomen, while gastric distention may be symptomatically represented by marked pulmonary symptoms. Cerebral symptoms which would at a later period point towards organic lesions of the brain or its meninges, often in early life simply indicate a disturbance of the meningeal circulation which may arise in almost any disease with a heightened temperature or with interference with the circulation, as in pertussis, in cardiac disturbance, and in acute gastro-enteric disease. The reverse of this picture is at times seen in cases of considerable pleuritic effusion with displacement of the heart, in which very slight rational signs, in comparison with the same condition in adults, are shown, the child appearing fairly well and inclined to run about its nursery. Again, with very slight lesions in the lungs, most violent pulmonary symptoms may arise. An insignificant disturbance of digestion may present most alarming symptoms of pallor and collapse. Infants and young children may, from purely nervous causes, vomit so continuously that the symptoms, when the vomiting has ceased, closely simulate a typhoid condition, and, still more frequently, tubercular meningitis.

The prodromal symptoms of many diseases, both benign and malignant, simulate each other so closely in early life that the diagnosis must often be left in abeyance for many days longer than would be necessary at a later period. Pulmonary, gastro-enteric, and cerebral diseases frequently show this correspondence of symptoms. Affections of the ear, where the aural symptoms are marked, as they often are, produce a great variety of symptoms pointing towards other organs, such as cough and vomiting, and owing to the late closure of the petrosquamosal suture, the most pronounced cerebral symptoms. The rational indications of a disease, therefore, in early life are a symptom-complex, and we have to rely almost entirely on our physical examination. It must be remembered, however, that seemingly grave and alarming symptoms do not necessarily indicate a serious disease, while mild and unobtrusive symptoms may be the beginning of a fatal disease.

Of especial importance in infantile symptomatology are the cry, the facial expression, the posture, and the movements of the body; these will be described when the various diseases are spoken of.

GENERAL DIAGNOSIS.—It has already been stated that the diagnosis in children is most satisfactorily made by the physical signs, but the examination is often so difficult to obtain that we necessarily also depend greatly on the history given by the parents and on inspection. There are, however, certain methods of examination which are preferable to others and which differ in their system from those which we employ in adults. The employment of these methods, though involving more time, render it possible to obtain a satisfactory examination in almost every case.

GENERAL PROGNOSIS.—The mortality in early life is greater in inverse proportion to the age, so that the prognosis in diseases which in older children would be regarded as benign should in the early weeks and months of life be considered as serious, and the prognosis should be correspondingly grave. On the other hand, certain organic diseases, such as those of the kidney and heart, which would be attended with a very bad prognosis in later life, in childhood are much more likely to recover, on account of the wonderful recuperative powers of children due to their rapid reconstructive metabolism.

Young children may die after a few hours' illness apparently from their lack of power to resist the onset of various infections, such as scarlet fever and pneumonia, before these diseases have declared themselves by their characteristic symptoms. Sudden death from various and obscure causes is also not very uncommon in weak infants who are suffering from malnutrition. Internal hemorrhages, as in Buhl's disease, asphyxia from many causes, sudden collapse in the course of pertussis, laryngospasm, or cardiac disease, may also be the cause of sudden death.

MATERNAL IMPRESSIONS.—A few words should be said concerning the subject of maternal impressions. For many years there has been accumulating a considerable amount of evidence showing that a violent mental impression made upon a woman who is at the time carrying a child may be followed by a physical or mental defect in the child which bears a striking relation in character to the impression made upon the mother. Thus, Sir Walter Scott narrates that King James the First could not endure the sight of a drawn sword. This feeling had been attributed by those who believe in maternal impressions to the terror which his mother experienced at witnessing the murder of Rizzio. Still more numerous are the facts adduced to prove that bodily defects, such as harelip, club-foot, and hairy mole, may be caused by strong impressions of pain or terror experienced by the mother at the time when the fœtus is in a certain stage of intra-uterine development. Interesting as these instances are, it is the general belief that nothing more has been proved than that they depend on a coincidence. The final decision on this obscure subject must rest on future investigation, and until something more definite is known we should guard a woman during her pregnancy from all unpleasant impressions with far more care than we do at present.

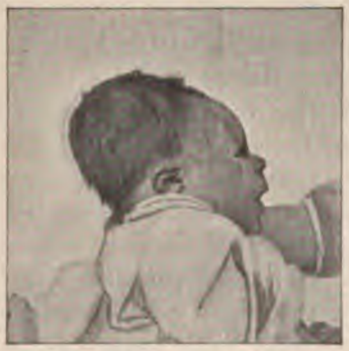
The diseases which we speak of as *diseases of the new-born* are distinct from those which are acquired later in life, in that they represent in almost every case an arrest of the normal development which should occur during intra-uterine life. A stage of development which is normal at a certain period of intra-uterine life becomes abnormal if it persists to a later period, and this persistence of an early stage of development constitutes in the great majority of cases what is known as congenital malformation. Such a failure of development may be the result of intra-uterine inflammation, which, either by crippling the various functions or by arresting the normal intra-uterine growth, produces a condition of disease at birth. In many cases, however, the causes are so obscure as to elude our usual methods of examination. Diseases of the new-born may also be made to include certain abnormal conditions which arise immediately after birth or in the early days of life.

Although many of these affections must pass into the hands of the surgeon for treatment, yet it is very important for the medical practitioner to be able to recognize at once their true nature and their significance. For purposes of simplicity, these diseases can be classified into *diseases of the head*, *diseases of the neck*, *diseases of the trunk*, *diseases of the extremities*, and *general diseases*.

DISEASES OF THE HEAD.

Caput Succedaneum.—The normal average head at birth may be misshapen from various causes. Of the conditions which may cause unusual appearances, the most common is called *caput succedaneum*, a case of which is represented in Fig. 70.

Fig. 70.



Caput succedaneum. Male, 2 hours old.

This infant, a male, two hours old, presented a swelling over the right parietal bone extending back to the occiput, causing an irregular tumor and a great increase in the anteroposterior diameter of the head. The tumor did not fluctuate. The presentation was occiput left anterior, and no instruments were used. The swelling corresponded to the place where there was the least pressure,—that is, the presenting part.

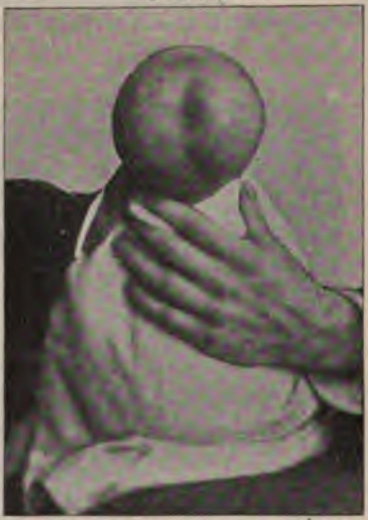
It is needless to say that this caput succedaneum requires no treatment, as it gradually disappears of itself by absorption in a few days. It is simply a swelling of the scalp caused by a passive congestion with extravasation of blood and lymph into the connective tissue external to the pericranium.

Caput succedaneum must be carefully distinguished from another swelling of the scalp, *cephalhæmatoma*, which may occur in connection with it, and which appears as the caput succedaneum disappears.

Cephalhæmatoma.—During labor a hemorrhage may take place from the blood-vessels of the head which gives rise to a tumor in one of three situations: (1) between the occipito-frontalis aponeurosis and the periosteum; (2) between the periosteum and the skull; or (3) between the skull and the dura mater. The first two are known as *external cephalhæmatoma*, the last as *internal cephalhæmatoma*. The cause cannot be entirely from pressure over the presenting part, as the lesions have been found in breech presentations.

Cephalhæmatoma is distinguished from caput succedaneum by its sharp limitation to one of the parietal bones, by its fluctuation, and, if seen late, by its surrounding bony wall. It can be diagnosticated positively by the withdrawal of some of the fluid by a hypodermic syringe. Another condition which may simulate it somewhat is a depressed fracture. The differential diagnosis from this latter condition can best be made by remembering the fact that the resistant rim of the cephalhæmatoma is raised above the level of the surrounding bone, and is somewhat compressible, while on the inside it can be felt to slope evenly towards a fluctuating centre. In fracture no such arrangement occurs.

FIG. 71.

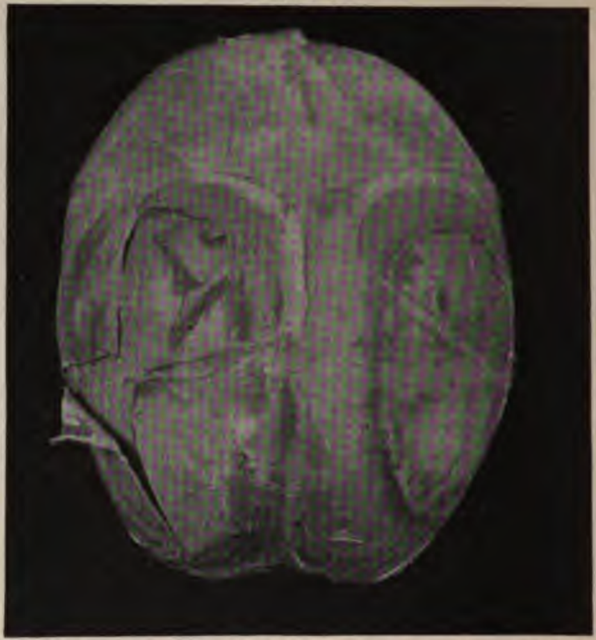


Double cephalhæmatoma. Infant, 4 days old.

(*a*) *External Cephalhæmatoma.*—By far the most common form is that in which the tumor has formed between the skull and the periosteum. It shows itself as an irregular circular swelling over a parietal bone, and gives on palpation a distinct feeling of fluctuation. The skin over it is not discolored or reddened. In the cases that have existed for a few days a bony wall can be felt surrounding the tumor, the edges of which give a crackling sensation under the finger. In this stage it may strongly suggest a fluid tumor coming through a circular hole in the skull.

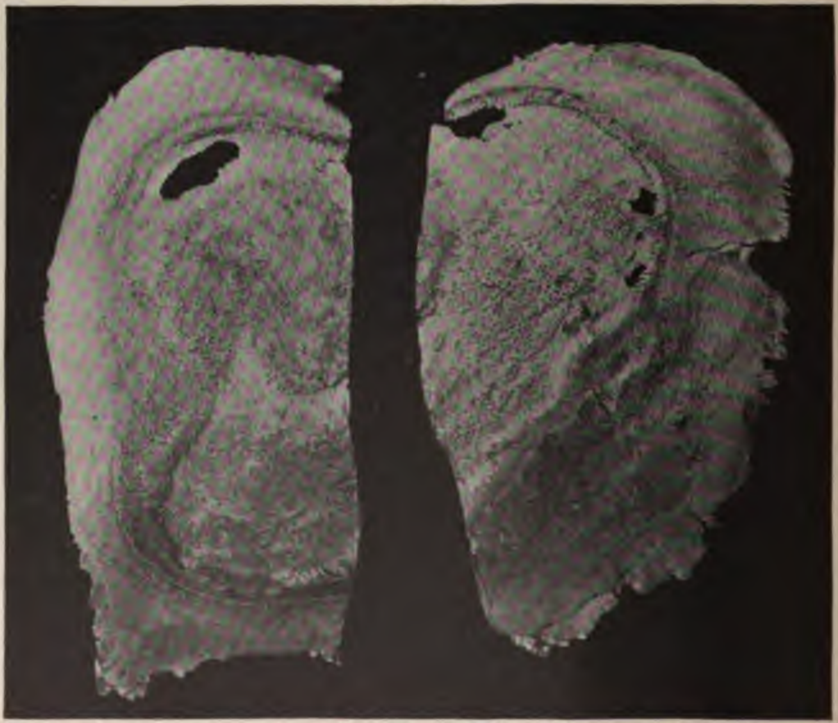
Fig. 71 represents a case of double cephalhæmatoma of the external variety; that is, it is an extravasation of blood under the pericranium. Its base corresponded to the denuded bone, and was oval or circular. There were bulging tumors on each side of the sagittal suture with a deep sulcus between them. On palpation there was fluctuation, and on feeling the circumference of the tumor there was noticed an elevation and a crackling sensation as though one were touching fine crystals of ice on the edge of water which is beginning to freeze.

FIG. 72.



Double external cephalhæmatoma. Both parietal bones. Warren Museum, Harvard University.

FIG. 73.



External cephalhæmatoma. Parietal bone dissected. Warren Museum, Harvard University.

2 represents the dried preparation of a double cephalhæma- h is in the Warren Museum.

left side of the skull (the right side of the picture) the integu- been nearly removed, showing a raised bony rim.

right side of the skull (the left side of the picture) the integu- been cut off and partially deflected, showing the cavity which the diffused blood.

3 is a parietal bone dissected so as to show the condition of n a case of external cephalhæmatoma.

ecimen shows well the raised rim and the porous condition of underlying the tumor. In two or three places the bone sub- entirely disappeared.

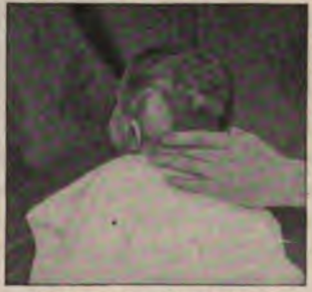
ternal Cephalhæmatoma.—Internal cephalhæmatoma is situated he inner surface of the skull and the dura mater, and is rare. es found in connection with the external variety.

rognosis in these cases is bad. They are usually fatal, and there wn treatment which can save them.

gocele.—By the term meningocele is understood a protrusion art of the membranes of the brain through a hole left in the ll by defective ossification. In some instances this is caused by terine hydrocephalus. These tumors generally contain some bro-spinal fluid in the bag of membrane. Such fluid can often d into the skull by gentle pressure, but at the risk of bringing on of cerebral disturbance.

represents a small meningocele above the left ear about 2.5 cm. (1 inch) Some fluid was withdrawn from it by an aspirating needle, and the con- sac proved to be serous without cells. The sac refilled after tapping. No ive operation on it has so far been undertaken. The child was rhachitic. some time previous and struck its head. Nothing abnormal was noticed hild previous to the fall, but after the ac- lling appeared above and behind the ear. increased in size when the child cried, ctuating, and not tender. The knee-jerks on were normal. The ophthalmoscopic disclosed nothing abnormal.

FIG. 74.



Meningocele. Female, 3 years old.

h more serious condition is shown ingocele in the following case.

ant was two weeks old when operated ind its left ear was an irregular tumor n. (3 inches) long. The ear was pushed appeared to be growing from the tumor. as normal, and the infant at birth was lthy and well formed, except for the tumor, which was congenital. On the tumor was found to be fluctuating and translucent. There were large urface. No impulse could be felt on crying, nor did pressure cause any ptoms. On aspirating it, 45 c.c. ($1\frac{1}{2}$ ounces) of a clear reddish fluid were withdrawn. This fluid contained red blood-corpuscles and a few endothelial cell unfavorable symptoms followed the aspiration. After the withdrawal of the flu openings could be felt, the anterior probably connecting with the external au meatus and the posterior with the anterior fontanelle. The tumor was increas size so rapidly that an operation was decided upon. On removing it an openi the skull large enough to admit two fingers was found. The child made a rap covery from the operation, and was left with only a scar behind the ear. There no cerebral symptoms. During convalescence the child seemed to be mentally b

Encephalocele.—Still more common than the pure meningoce that condition in which the hernia contains some of the cerebral stance as well as the membranes. This condition is called encephaloc or if, as is often the case, it contains a portion of a dilated ventricle, so the tumor is filled with cerebro-spinal fluid, it is known as hydro-ence locele or as hydro-encephalo-meningocele.

Fig. 75 represents a remarkable case of hydro-encephalocele wh was treated by Dr. Lovett in the hospital.

The infant from the time of its birth had tonic and clonic convulsions, occurr usually as often as once in three hours. It was brought to the hospital when it two months old. It was well formed in every way, except that it had a tumor on back of its head which was at least one-third as large as its skull. The tumor only partly covered with skin, the upper part of it being a thin translucent membra It communicated with the brain through a large square hole in the back of the sku The tumor fluctuated slightly and appeared to be a multilocular cyst, for when it w aspirated only a part of the contained fluid could be withdrawn. The tumor w removed by Dr. Lovett and the wound sewed up tightly. The cyst was found to co tain a viscous fluid with slight flakes in it which proved to be particles of cerebr substance. The convulsions immediately became less frequent, and ultimately treatment with bromide of potash disappeared almost entirely.

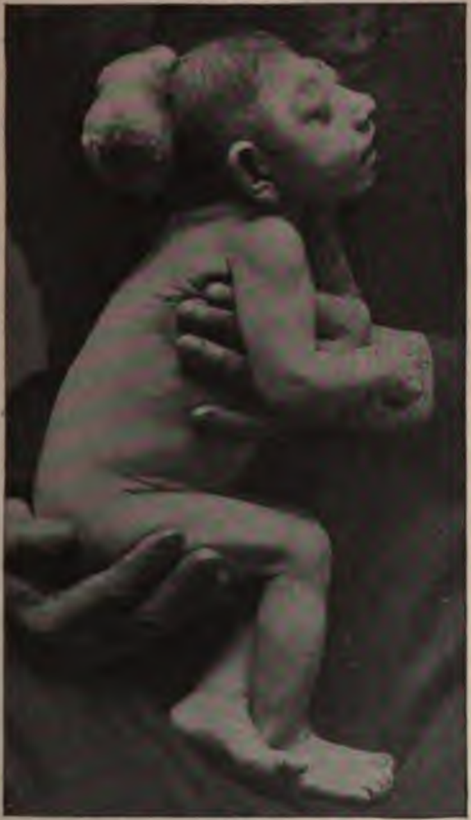
The infant in other respects was very little affected by the operation, and reco ered rapidly. After remaining in the hospital two weeks it was taken to its home where it died some months later of some intercurrent affection.

Regarding these tumors in general, it is enough to say that we should view with suspicion any fluctuating swelling that seems to have a deep attachment in the neighborhood of one of the cranial sutures. The most frequent seat of these tumors is in the occipital region and at the root of the nose. Their treatment has not proved very successful. Some few may steadily decrease of themselves and ossification may block up the abnormal opening. Pressure and the injection of Morton's fluid have both been tried, and in some cases have been attended with success. At present the operative plan of treatment is considered the best. Without interference the tendency is usually towards rupture of the hernia, convulsions, and death.

Anencephalia.—The cerebro-spinal system is formed from the medullary tube, which is made by the infolding of epiblast along the medullary groove: if the formation of the medullary tube is for any reason incomplete, or if the dorsal wall of the tube is destroyed, the cerebrum or part cerebral axis will remain rudimentary. According to the amount ference with the development we may find more or less of the emaining in a rudimentary condition, and thus producing greater degrees of what is called anencephalia. Total anencephalia is Partial anencephalia is much more common. These cases are not cial interest, as it is exceptional for them to live beyond a few days.

igenital Hydrocephalus.—One of the more common malforma- f the head is a hydrocephalic condition at birth, called congenital ephalus. It is described on page 970.

Fig. 75.

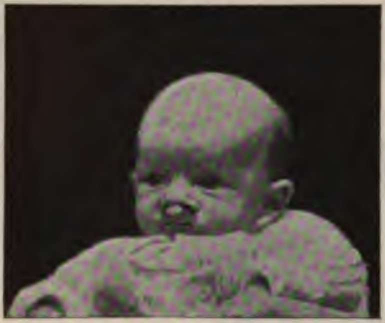


Female, 2 months old. Hydro-encephalocele.

—If the maxillary process on one or both sides of the face with the intermaxillary process, a cleft will remain open in f the upper lip on one or both sides of the intermaxillary nce we shall have single or double harelip as the case may eft extends the whole distance from mouth to nostril it is te, but if the nostril is not reached by the opening it is called If there is a failure of the palatine processes to join, one s will open into the roof of the mouth as well as into the pharynx, and we shall have the malformation known as cleft palate. This may be a large chasm running the whole length of the roof of the mouth, or may be only a small opening, or nothing but a bifurcation of the tip of the uvula may be left to show that the normal process of development has not gone on to completion.

Besides their unsightly appearance, which always causes the mother great concern, these malformations may so interfere with the infant's taking the breast as to render sucking impossible and make it necessary to feed the infant with a spoon.

FIG. 76



Double harelip.

Fig. 76 represents a typical case of double harelip uncomplicated by cleft palate.

The intermaxillary bone was of a large size, protruded considerably beyond the margin of the lips, and was somewhat twisted upon itself. This alteration of the position of the intermaxillary bone may cause the teeth that grow from it to appear in very unusual places, so as to protrude, for instance, from the nostril.

The operation should be performed during the early weeks of life, as the growth of the facial muscles is not then sufficient to interfere with the healing of the wound.

There is considerable difference of opinion as to when cases of congenital harelip should be operated on. In general, it can be said that cases of single harelip unassociated with cleft palate can be operated on from three to six weeks after birth, while the severer forms are best left for as many months. When the infant is wasted and is in a poor general condition, the indication is to postpone the operation, as it is seldom that this defect is the cause of the general lack of proper development.

Cleft Palate.—In speaking of harelip most of the conditions occurring in cleft palate have been described. The difficulty of feeding, if the cleft involves the hard as well as the soft palate, is very great, and can best be accomplished with a spoon. The difficulty in articulation and the unpleasant sound of the voice are reasons which lead the parents to demand early treatment. We should wait a longer time before operating than in cases of harelip, as it is seldom wise to operate upon this deformity before the child is three years old. The operation for cleft of the soft palate is called staphylorrhaphy, and that for the closure of a cleft in the hard palate is termed uranoplasty. The larger the opening in the palate the more successful will be the treatment by apparatus in comparison with that by the knife, and many prefer to close the cleft by fitting artificial plates.

Tongue-Tie.—In quite a number of cases the frænum of the tongue is abnormally short at birth. In extreme cases the tip of the tongue is so closely bound to the lower jaw that it cannot be protruded beyond the line of the gum or touched to the roof of the mouth. The mother usually notices that the infant does not nurse readily, and brings it to the physician to discover the cause. In most cases on passing the finger into its mouth the infant is found to suck fairly well; but there can be no doubt that this condition, which is called tongue-tie, interferes somewhat with the process of sucking.

Children who have not learned to talk at the usual time in the second and third years are frequently brought to me with the statement that they are tongue-tied, and the parents wish the condition to be treated. Large numbers of children are taken to the physician under this supposition but in very few instances are they tongue-tied. These children belong to the class of retarded speech. The condition is a central one of the brain, and not a local one in the mouth, and if children hear well and are bright and mentally well developed, even though they do not speak at the third, fourth, or even fifth year, as a rule they learn to speak later.

TREATMENT.—The treatment is to cut the frænum. This operation should be followed by no hemorrhage and requires no dressing. Having the child's head held in a fairly good light by an assistant, and guarding the lower part of the tongue with the perforated flange of a director, a small cut is made in the tense frænum with a pair of blunt-pointed scissors. By making the cut close to the gum there is no danger of wounding the ranine artery. The cut is prolonged as far as is necessary by tearing with the finger-nail.

Ranula.—Beneath the tongue we sometimes find the mucous membrane bulging out as a bluish, translucent tumor which is soft, painless, and semi-fluctuating. This condition is called *ranula*, and is a retention cyst caused by the blocking of a mucous duct. When opened, a small amount of glairy fluid escapes, but the collapse of the walls of the cyst brings the edges of the cut together and they quickly adhere. The fluid will soon re-collect; therefore the only sure way of dealing with these cysts is to pinch up their anterior wall with fine forceps, and with the scissors remove so much of it as to leave no opportunity for the edges to adhere. A gentle application of nitrate of silver to the edges and interior of the sac after the cut has been made with the scissors materially helps to promote the cure. It is not common in new-born children, but it occurs often enough to deserve mention.

Protrusion of the Ears.—A deformity which is quite frequent at birth, and which increases as the infant approaches childhood, is the protrusion of the ears. The ear, besides at times being placed in an irregular position on the head, has in these cases a tendency to stand out from the head farther than is considered normal. This position of the ear usually annoys a mother very much, and the physician will frequently be consulted as to the means by which the deformity may be rectified.

TREATMENT.—In a large number of cases the persistent applicat pressure by means of various devices, one of which is a fenestrate will cause the ears to be flattened against the side of the head. In ir able cases an operation will have to be performed, but it is very s and does not leave an unsightly scar.

Ophthalmia Neonatorum.—Ophthalmia neonatorum has been di into two forms, the catarrhal and the purulent.

(*a*) *Catarrhal Ophthalmia.*—The catarrhal form may be caused b slight irritation of the eyes of the infant. It runs a very mild cours inflammation attacking chiefly the palpebral conjunctiva. Often the symptoms noticed are a slight photophobia and a collection of the se tion in the angles of the lids and upon their borders. Its whole cour mild, and often it is all over in a few days.

(*b*) *Purulent Ophthalmia.*—Although a considerable number of ca for purulent ophthalmia in the new-born have been given, such as trau exposure to light and cold, and others, certainly ninety-five per cent. all cases are caused by infectious material from the genito-urinary t of the mother, and in most instances by gonorrhœal pus. The ea signs of the disease may appear at any time from the third hour of l and the earlier the pus appears the more virulent will be the course the disease and the more unfavorable the prognosis. If infection tak place during the birth of the child, the symptoms usually begin on t third day; but, as contaminated linen and fingers may carry the infectio material to the infant's eyes at a later period, the symptoms may l delayed indefinitely.

SYMPTOMS.—The disease begins as a redness of the conjunctiva, wit a slight discharge from the corner of the eye. This is succeeded wit startling rapidity by intense inflammation of the lids. In twenty-fou hours the upper lid may become so much swollen as to overhang th cheek and render opening the eye impossible. On separating the lids, little greenish pus, which may even be tinged with blood, wells up betweer them. At first the cornea is unaffected, but if the pus accumulates unde the œdematous lids it soon shows signs of ulceration. In the second twenty-four hours the ulceration may perforate the cornea and evacuate the aqueous humor, thus bringing the iris into contact with the posterior surface of the cornea. The inflammation may extend around the eye and well over the forehead and malar prominence, but it does not persist in the latter region very long. All the symptoms disappear slowly, and recovery takes place, except in those cases in which the cornea has been permanently injured by ulceration.

TREATMENT.—In treating this disease we must be very prompt and energetic. It often may be averted by what is known as Credé's method. This consists in dropping one or two minims of a two per cent. solution of nitrate of silver into each eye of the new-born infant. Although this has been known to cause even a considerable amount of irritation, yet it undoubtedly exerts a powerful influence in warding off this dangerous disease.

After the disease has once begun, two indications must be kept in mind: (1) to reduce the inflammation, and (2) to prevent the pus from accumulating behind the tightly closed lids. By far the best way of applying cold to the eye is by compresses of thin, soft pieces of linen cut into small squares. Not more than two thicknesses are to be used at once. These compresses are to be cooled by laying them on a piece of ice or floating them in ice-water. They must be constantly changed. To remove the pus, a gentle irrigation, such as can be easily obtained by using a medicine dropper, is sufficient.

The secretion is highly contagious, not only for the infant's other eye, but for those who are taking care of it. Therefore one must avoid all spattering, and should cover the infant's well eye before beginning the irrigation.

In the irrigation of the eye one should first turn the child's head a little to the diseased side, and with the fingers of the left hand gently separate the lids as far as possible. Then, holding the dropper with the right hand, irrigate between the lids, directing the stream *from* the nose. After each irrigation vaseline should be applied to the edge of the lids. This should be done at least every half-hour, day and night, until the swelling has so far subsided as to preclude the danger of any secretion being retained. For irrigation many solutions have been advocated. The most simple, and perhaps the best, is a saturated solution of boracic acid, or one of bichloride of mercury in the strength of 0.05 gramme (1 grain) to 480 c.c. (1 pint) of distilled water. In the later stages of the disease, where all the tissues are relaxed, a solution of nitrate of silver, 0.5 gramme (10 grains) to 30 c.c. (1 ounce) of distilled water, may cautiously be used once a day. This same solution painted on the conjunctiva with a camel's-hair brush once daily early in the disease is very effective in shortening the course of the disease.

DISEASES OF THE NECK.

Hæmatoma of the Sterno-cleido-mastoid Muscle.—During the birth of the child, either from the violence of the expulsive efforts of the uterus, or, as more frequently happens, from the pressure of the forceps in head presentations, or from too vigorous traction upon the feet in breech presentations, or for no assignable reason, the sterno-mastoid muscle may be partially ruptured in its sheath and a hæmatoma form between the torn ends. This tumor may be either in the sternal or in the clavicular portion of the muscle, or may be just above the junction of the two. For a short time it is soft and tender, but gradually it loses its sensitiveness and becomes converted into fibrous tissue, which then tends to contract. It may appear as a small tumor, but in infants with fat necks it may not be noticeable at first. As turning the head towards the affected side lessens the tension upon the swelling, the infant will rigidly hold its head in that position. It is in this way that cases of infantile torticollis are thought by most writers to arise.

Treatment.—After the painful stage has passed, the treatment is by gentle massage and manipulations addressed to stretching the shortene muscle. If these methods fail, the child must be placed in the hands o an orthopædic surgeon for more extended treatment, either by apparatus or by division of the tendinous attachments of the sterno-mastoi muscle.

Branchial Fistulæ.—At an early period of development the neck o the fœtus has along its sides a series of four branchial clefts, which communicate freely with the œsophagus and represent the gills of aquat animals. The upper one of these forms the tympanum and the eustachia tube, the rest are normally obliterated. Sometimes we find traces o these branchial clefts in the form of small fistulous tracts which admit probe a short distance and end blindly. Their most frequent seat just above the sterno-clavicular articulation, but they may be found anywhere along the anterior border of the sterno-mastoid muscle. Sometimes the entrance of these fistulæ becomes stopped, so that they dilate and form large cysts containing mucus, blood, and atheromatous detritus. These form at times large and unsightly bunches, which require surgical treatment. Often the operation of obliterating them is not an easy one, for they are apt to have deep and complicated attachments.

Treatment.—If they do not cause any inconvenience it is better to let them alone, as they often prove very intractable to treatment. If they are annoying because of a slight mucous discharge, we can try to eradicate them with the galvano-cautery, or by passing a probe into the wound and dissecting from around it the lining of the sinus.

DISEASES OF THE TRUNK.

Mastitis.—In certain infants during the early days of life we find a swelling and hardness of one of the mammæ. This condition appears to be an inflammatory one, and is abnormal. In connection with the swollen condition of the mamma, a secretion is found to come from the nipple which corresponds closely to milk, and which has been called "witches' milk."

A number of analyses have been made of this fluid, and the following are some of them:

(Schlossberger.)

Fat	0.82
Casein, sugar, and extractives	2.83
Mineral matter	0.05
Total solids	3.70
Water	96.30
	100.00

(Von Gesner.)

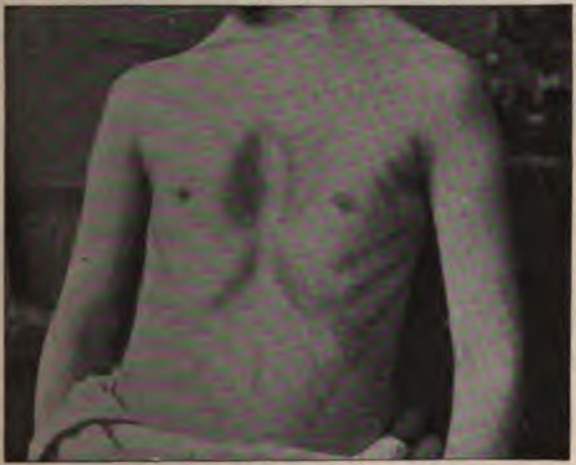
Fat	1.45
Casein	0.55
Proteids	0.49
Sugar	0.95
Mineral matter	0.82
Total solids	4.26
Water	95.74
	100.00

This condition occurs in boys as well as in girls, and, as far as I know, has no special significance. With ordinary antiseptic precautions the inflammation usually subsides in a few days, leaving the affected breast the same size as the other. The following case represents this condition of the mamma in a female one week old:

The swelling of the mamma was noticed on the fourth day of her life. A little fluid looking like diluted milk could be expressed from the mamma. The treatment of the case was simply to keep it thoroughly clean by washing it with sterilized water, carefully drying it, and applying a compress with a little simple ointment on it.

Depressed Sternum.—There are a great many congenital malformations which may occur in different parts of the thorax. Fig. 77 represents one of these. It occurred in a boy who was born with a depression of the lower part of the sternum.

FIG. 77



Congenital depression of sternum. Male, 6 years old.

He was six years old when first seen, and had a rounded depression, about 4 cm. (1½ inches) in diameter, beginning at the third costal cartilage and extending to the ensiform cartilage. He was perfectly healthy. The cardiac dulness extended to 2.5 cm. (1 inch) to the left of the mammary line, and its impulse was in the fourth left interspace. The spinal column was straight. The epiphyses of the wrists were slightly enlarged, but there was no other evidence of rhachitis. When he was two months old he had a severe attack of pertussis, which lasted for over two months. At five years of age he had a very severe attack of bronchitis.

Although this depression of the sternum was present at birth, and later increased in depth and in circumference, it eventually ceased to enlarge. The circumference of his head and that of his chest was 50.5 cm. (20 inches). The heart was somewhat displaced upward and to the left, but was apparently unaffected by its abnormal position. Light gymnastic exercises to broaden the chest and to strengthen the thoracic muscles were advised for treatment. Such a malformation as this sometimes results as one of the changes subsequent to Pott's disease. More often the sternum protrudes, but occasionally recession takes place, closely resembling the condition in this case.

Prominent Sternum.—A prominence of the sternum, called *pigeon breast*, occurs more often than the depression. It may happen without a assignable cause, or it may be due to rhachitis, and may also result from some spinal distortion, such as that of Pott's disease, or lateral curvature. In the latter case the sternum is often tilted to one side.

Spina Bifida.—Spina bifida consists of a lack of closure of the laminæ of the vertebræ. This condition is normal at a certain period of intra-uterine life, but when persisting to a later period, and when occurring at birth, becomes abnormal from a developmental point of view and represents a distinct malformation. As the fusion of the laminæ at the base of the spinous process takes place in sequence from above downward, the most frequent seat for spina bifida is in the lumbar and lumbo-sacral regions. There it appears as a tumor situated exactly in the middle line, covered sometimes with healthy skin, but as frequently roofed over by nothing but a thin adherent transparent membrane. Rarely the tumor is solid, containing nothing but an empty sac that has been walled off from its connections with the spinal canal. It is then called *spina bifida occulta*. In true spina bifida the tumor is filled with cerebro-spinal fluid, which can be seen to increase in amount as the child cries, and can, by pressure upon the sac, be forced back, in this case often giving rise to cerebral symptoms. According to the contents of the tumor, spina bifida has been divided into several varieties.

(*a*) *Spinal Meningocele.*—When there is a protrusion of the membranes filled with fluid the tumor is called a spinal meningocele.

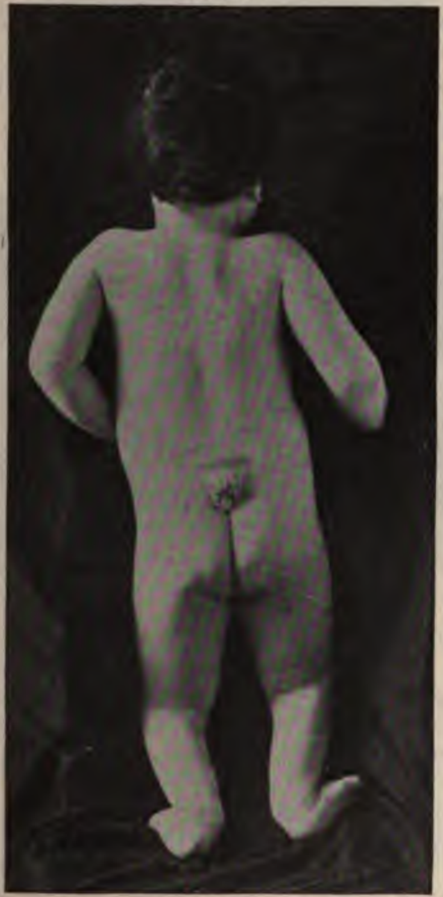
(*b*) *Meningo-myelocele.*—The most common form is where the spinal cord, as well as the membranes, is found in the tumor. It then becomes a meningo-myelocele. The position of the cord in these tumors is a very variable one. It may run directly through the tumor and even be suspended by a kind of mesentery; or, as is usually the case, it may be spread out like a fan over the surface; in any instance it is rudimentary in character.

(*c*) *Syringo-myelocele.*—Syringo-myelocele is a rare form, in which the sac is formed of meninges and cord, the central canal of the cord being dilated to make the cavity of the tumor.

Spina bifida occurs usually in poorly developed infants, and in a large majority of cases it is associated with other malformations, such as congenital hydrocephalus, harelip, club-foot, paralysis of the lower ex-remities, and in severe cases there may be incontinence of urine and of eces. Sometimes the infant is well formed and healthy in every other spect.

If left to itself, the course of spina bifida is in one of two directions:) spontaneous closure and obliteration of the sac; (2) ulceration of the c, usually followed by convulsions and death. In the first case, which very rare, the sac shrivels up and thus effects a spontaneous cure. The lowing case was one of spontaneous closure:

FIG. 78.



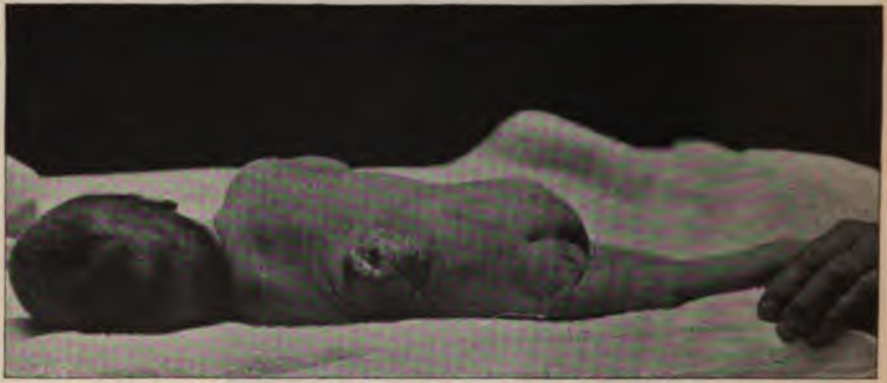
Spina bifida. Spontaneous cure. Male, 4½ years old.

A boy, four and one-half years old, showed an elevated cicatrix in the lumbar gion, which suggested the former existence of a spina bifida. The case was seen y Dr. Lovett when it was eighteen months old, and so far as could be learned there ad been a large tumor present at birth. The sac burst in this case, and, contrary the general result, the child did not die, but was left with paralysis of the legs, hich made it stand in the curious and abnormal position shown in Fig. 78. He also suffered from incontinence of urine and of fæces. The child had never walked, and it seemed probable that his disability was caused by the fact that the nerves were spread on the walls of the sac, as is usual in many cases, and that they were incorporated in the cicatrix.

A result such as is described in the above case is, however, very exceptional. The rule is, either that there is an ulceration of the sac, followed by a large loss of cerebro-spinal fluid, convulsions, and death, or that the opening in the spine being very small the loss of fluid is constant, and the result is the same. In some instances there is an infection of pyogenic organisms through the walls of the sac, which causes a septic meningitis in the cord, and finally in the brain. Such a case has been reported and beautifully illustrated by Holt, showing the presence of the bacteria and a resulting purulent hydrocephalus.

Fig. 79 represents another case of spina bifida which will illustrate the ordinary course of the affection.

FIG. 79.



Spina bifida of dorsal lumbar region. Infant 48 hours old. Died when 10 days old.

It shows a large spina bifida in the dorso-lumbar region. The membrane covering the tumor was so translucent that the spinal cord could be plainly seen through it. At birth there was a small tumor. It filled with fluid at the end of twelve hours, and at the end of forty-eight hours it looked as it does in this picture. The top of the tumor suppurated, the fluid began to leak away, and the child died within ten days.

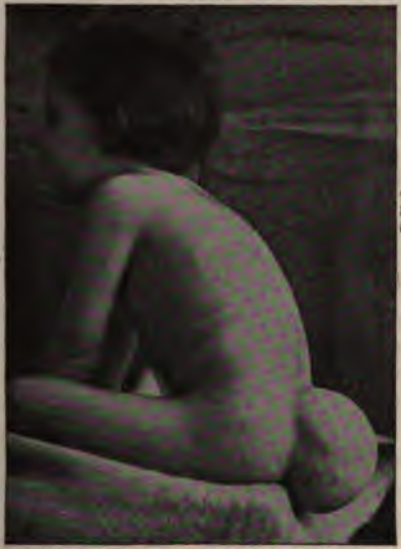
This is the course pursued by the disease in the majority of cases which are not operated upon.

Fig. 80 represents the case of a boy five years old, who had had this large tumor since birth. It was situated over the lumbar region of the spinal cord, and in the median line. The fluid was withdrawn several times for purposes of examination, and when the sac was lax an opening 5 cm. (2 inches) long could be felt in the spinal canal. It was elliptical in shape. From the fact that the child suffered from incontinence of urine and had a certain degree of paralysis of the legs, it was fair to infer that the nerve-supply of the legs and pelvis was incorporated in the tumor.

This case was tapped and treated with an injection of Morton's fluid, but the treatment was entirely unsuccessful, and although the sac was aspirated several times the fluid always returned.

TREATMENT.—The only rational treatment of spina bifida is by operation, and the operation now commonly done is excision of the sac and closure of its neck.

FIG. 80.



Spina bifida of lumbar region. Male, 5 years old.

Rhachischisis.—Rhachischisis is one of the principal forms of congenital defects of the spine. It is characterized by a deficiency of the vertebral arches either complete or partial. The cord is rudimentary and is split open so that the endothelial lining of the central canal is exposed. This may occur in the whole of the cord or in a part of it, constituting total or partial rhachischisis. This disease is of pathological rather than clinical interest, as the infants die in a short time.

Phlebitis and Arteritis Umbilicalis.—The cause of both of these conditions is a septic infection of the umbilical stump. It is considered by most pathologists to begin as an inflammation of the perivascular cellular tissue, and only secondarily to invade the walls of the vessels. The region around the umbilicus is red and hot, and we may be able by gentle pressure to squeeze a few drops of pus from the stump of the cord. It is a very dangerous affection, as septic emboli readily pass from the infected vessels into the general circulation and set up metastatic inflammation in the thoracic as well as in the abdominal organs.

TREATMENT.—The treatment is to sustain the infant's vitality by stimulation and thoroughly to disinfect the umbilicus with solutions of bichloride of mercury or carbolic acid, followed by the application of boracic acid or iodoform powder. A flaxseed poultice is often of service, and some authors recommend placing the infant upon its abdomen in order gravity may aid in draining away the pus.

Congenital Umbilical Hernia into the Cord.—Dr. Howard Mars the Report of St. Bartholomew's Hospital for 1874, calls attention to "familiar anatomical fact that from about the sixth to the twelfth w of intra-uterine life the cæcum and neighboring portions of the ileum contained in the part of the umbilical cord which is next to the body the embryo, and that they should subsequently withdraw into the ca of the abdomen. In some cases, however, this recession fails to t place, and the intestine remains, even up to the time of birth, still lod in the beginning of the cord, which is dilated in the form of a memb nous sac." Not only may portions of the intestine be thus left outside the abdominal wall, but, as in a case recently operated upon by Warr the liver may be found lying in a hernial sac made from the dilated ba of the umbilical cord.

The infant was sent to Dr. Warren at the Massachusetts General Hospital a fe hours after its birth. At the umbilicus was seen the cord, which was greatly distend at its point of insertion into the abdomen, forming a tumor 6.5 cm. (2⅝ inches) i diameter. The coverings of the cord were inserted into a raised rim of skin, and we opaque, so that the contents of the hernia could not be determined.

When the infant was one day old, Dr. Warren enlarged the umbilical ring some what, separated the liver from the myxomatous tissue of the cord, which was in som places firmly adherent to it, and returned the mass within the abdomen. The woun was tightly closed with strong silk sutures. There was considerable shock following the operation, but there were no symptoms of peritonitis. In two weeks the wound had healed, and the infant recovered.

Fungus of the Umbilicus.—The umbilical cord, after being ligatured at birth, falls off by the seventh or eighth day, leaving a clean, dry cicatrix. After the separation of the cord we sometimes find a red protrusion, with a moist surface, that may even have a short central canal. This is generally due to an imperfect disintegration of the cord. It may bleed very readily if touched, and give rise to a discharge so irritating that the skin for some distance around the umbilicus becomes eczematous. This condition is called *fungus* or *polypus of the umbilicus.*

Treatment.—The treatment is very simple. The larger ones are best removed by ligation; the smaller ones can be destroyed by the application of nitrate of silver or the actual cautery.

Meckel's Diverticulum.—A condition which may at first simulate umbilical polypus, and of which umbilical polypus may be a symptom, is the persistence of a Meckel's diverticulum. This consists of the persistence of a piece of intestine, usually patent, connecting the small intestine with the umbilicus. It represents a vitelline duct that failed to atrophy when the placental circulation became established, and betrays its presence by an escape of fæces from the umbilicus. It is a rare malformation.

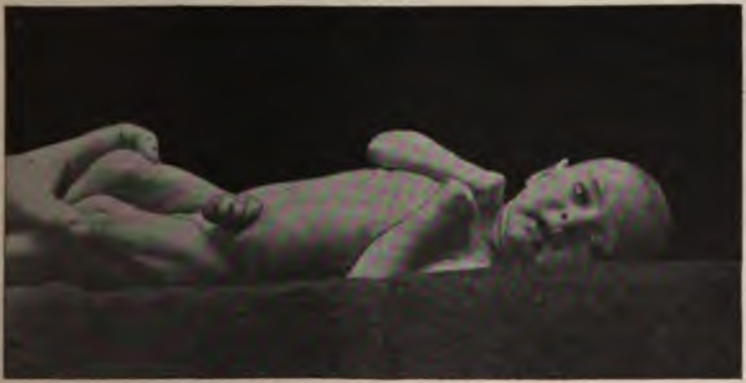
Umbilical Hernia.—The ordinary umbilical hernia, which is simply a protrusion of a knuckle of the intestines through the unclosed abdominal opening left by the separation of the cord, is of very common occurrence.

TREATMENT.—The lighter grades tend to recover spontaneously, and it is not advisable to operate upon them, or in fact on any umbilical hernia, until it has proved to be absolutely intractable, for it is an operation accompanied by considerable danger to the life of the infant. The lighter grades of umbilical hernia are usually easily reduced, but there is often great trouble in keeping them so. Various devices are employed for this purpose, but most of them are very unsatisfactory. At the Children's Hospital we are in the habit of proceeding in the following manner:

Having gently reduced the hernia, the skin of the abdomen is so pushed up between the fingers that it makes a vertical fold, at the bottom of which lies the umbilicus. The hole should be deep enough to lay one's finger in it. The tension is kept up by applying a wide strip of adhesive plaster transversely across the abdomen. This makes a pad of flesh, which closes the umbilical opening and retains the intestine in place. The cure is a slow one, and the treatment must be continued for many months in severe cases, without once allowing the hernia to come out. The milder cases are also aided by exercises which tend to develop the abdominal muscles. This can be very simply effected by having the child lie on the floor, and, while the feet are held down, making him rise to a sitting position with the back held straight. This is accomplished by the rectus muscles of the abdomen, and if the opening is a transverse one it tends to close it.

Fig. 81 represents an extreme case of umbilical hernia in an infant five months old. The hernia caused an eversion of the whole umbilical region.

FIG. 81.



Large umbilical hernia. Infant 5 months old.

Cases of incarcerated and even strangulated umbilical hernia have been reported, but are very rare. A few have been operated upon successfully. The danger from all such procedures is usually considered great, but there has been such an advance made in the modern methods of abdominal surgery that the operation is looked upon with increasing favor. Cases of hernia, whether umbilical or inguinal, are especially difficult to manage if the infant has pertussis or some similar disease.

Inguinal Hernia.—The most common forms of inguinal hernia that occur in young children are (1) the *congenital*, (2) the *funicular*, and (3) the *infantile* or *encysted*. An ordinary *acquired* form such as is the rule in the adult may be met with, but it is not so common. Strangulated and incarcerated herniæ occur at times, as in the adult, although they are rare.

There seems to be some evidence that the tendency to hernia is hereditary. Félizet reports eighty-five cases of hernia occurring in his practice, in which, omitting all cases in which the father pursued some laborious trade, such as that of a blacksmith, he found that in 24.7 per cent. the parents had had similar herniæ. Malgaigne reports a percentage of 29 due to heredity in a series of three hundred and sixteen cases of hernia.

Infants are at times brought to our hospitals with a history of colic who, on examination, are found to have more or less incarceration of these herniæ; which emphasizes the importance of making a systematic physical examination in every case for abdominal hernia, and of not taking it for granted that the symptoms are caused by indigestion.

(1) *Congenital Form.*—The congenital form is that variety in which the knot of intestine has made its way along a still patent funicular process. If it reaches into the scrotum it will be found completely to envelop the testicle.

(2) *Funicular Form.*—In the funicular form, the tunica vaginalis having become shut off from the funicular process just above the testicle, the hernia comes down the patent process, but does not envelop the testicle as in the preceding variety.

(3) *Infantile Form.*—Compared with the two forms just mentioned, the infantile or encysted form of hernia is quite rare, nor can it be diagnosticated with certainty without an operation. In it the funicular process has closed above but not below, and the intestine encased in a pouch of peritoneum forces its way into the process and descends.

DIAGNOSIS.—The diagnosis between direct and indirect hernia has little importance in childhood, as the inguinal canal is so short that the rings are practically at the same level. The condition which will be most readily confounded with hernia is hydrocele. Both give rise to an elastic tumor in the inguinal region and in the scrotum, and in fact resemble each other in many ways. Hydrocele is translucent by transmitted light; hernia is opaque. Hydrocele is always dull on percussion; hernia is usually resonant. If you can reduce them, hydrocele will go back slowly and noiselessly, hernia at the last quickly and with a gurgling sound. Hydrocele gives no impulse on coughing; hernia usually does. Lastly, in feeling for the inguinal ring in hernia it is found to be filled with the neck of the tumor; in hydrocele it is either empty or filled by a narrow stalk.

Treatment.—Most of the herniæ that will be met with in children are easily reducible, but we should remember that in attempting to get them back into the abdominal cavity we must use the greatest care, as nowhere can a little rough manipulation do more harm. If the hernia cannot be easily replaced, we should not think of leaving it where it is, simply because it gives rise to no alarming symptoms on the part of the child. No infant is safe with an irreducible hernia, and the sooner such a case is placed in the hands of a surgeon the better. Although the treatment of inguinal hernia, whether by actual operation or by the application of the usual trusses, should be in surgical hands, yet one method of treating these herniæ is so simple and safe that every medical man should know about it; in fact, in our children's clinics in Boston it is much used for all children under a year and a half. This method is the application of a worsted truss made of a skein of yarn which is passed under the back. The separated loop is around the side where the hernia is, and the unseparated end of the skein is brought forward and passed through the loop in the groin and up on to the back where it is fastened. In this way a soft slip-knot is made which lies directly over the inguinal ring and makes an excellent truss.

Femoral Hernia.—In femoral hernia the gut escapes from the pelvis under Poupart's ligament, and, making its way through the femoral canal, shows itself as a tumor directly under the saphenous opening. It can be diagnosticated at once from inguinal hernia by putting the finger on the spine of the pubes and noticing whether the origin of the tumor is to the outer or the inner side of that point. If outside, one is sure the hernia came through the femoral canal, no matter how far it may have extended up on to the abdomen. Femoral hernia is, however, extremely rare in young children, even in girls. In infancy the spine of the pubes, Poupart's ligament, and the anterior superior spine of the ilium are all much nearer together than in the adult. As a consequence, the femoral opening is so small and so well protected that it is usually impossible for the hernia to force its way through.

Hydrocele.—Several anatomical varieties are met with in hydrocele, as in hernia. Thus, if the collection of fluid occupies a freely open funicular process, we have the *congenital variety*, and the fluid can easily be returned to the abdominal cavity by placing the child on its back and elevating the scrotum. This is true also of *funicular hydrocele*, in which the fluid occupies an open funicular process, but is bounded below at the point where the tunica vaginalis has become walled off, leaving the testicle in a separate compartment underneath. When the funicular process has become walled off from the abdomen, but is still in communication with the tunica vaginalis, there may be a collection of fluid, which is then known as an *infantile hydrocele;* in this form the fluid is irreducible.

True hydrocele of the tunica vaginalis may be met with in children as well as in adults, but it is rare.

Encysted Hydrocele of the Cord.—There is another form of hydrocele which often escapes recognition, but perhaps still oftener is diagnosticated as hernia and treated with a truss. This is the encysted hydrocele of the cord. If in the course of the spermatic cord a hard, rounded swelling appears, and the testicle is found in its proper position in the scrotum and the inguinal ring clear, one is very surely dealing with a hydrocele of this kind. Having made the diagnosis, it should be evacuated with a fine aspirating needle. About 4 c.c. (1 drachm) of clear straw-colored fluid is generally drawn off, and the tumor usually disappears.

Encysted Hydrocele of the Canal of Nuck.—Analogous to hydrocele of the cord in boys is an accumulation of fluid in the canal of Nuck in girls. The appearance of the swelling is the same in both cases, and the treatment should be the same.

TREATMENT.—The treatment of all forms of irreducible hydrocele is first by aseptic evacuation of the fluid with a fine canula and trocar, or by an aspirating needle. If this, after repeated trials, fails to effect a cure, extirpation of the sac is the only sure method, although the injection of a weak solution of iodine is highly recommended by many authors. It is, however, dangerous in children, as the occasional connection of the hydrocele sac with the abdomen is not to be forgotten. Reducible forms of hydrocele are generally to be treated by a truss, in the same manner as herniæ, to try to effect a closure of the neck of the canal. If this is successful they can then be treated in the ordinary way. The outlook, however, is poor, and such treatment is generally unsatisfactory.

Undescended Testicle.—The testicle should descend into the scrotum at about the eighth month of intra-uterine life. In certain cases it does not descend, and if the descent does not take place within the first few years of life its function is lost from atrophy. It is, therefore, important in those cases in which the testicle descends and returns to the abdominal cavity to retain it in the scrotum by means of apparatus. Operation for this condition is not often successful. At times an undescended testicle is found in combination with an inguinal hernia. The following case of this kind came under my care about two years ago:

A boy, four years old, was discovered to have an inguinal hernia. The testicle was also found at times to be absent on the side of the hernia. Sometimes the hernia would descend and the testicle remain in the abdominal cavity, and again the testicle would come down with the hernia. It was exceedingly difficult to maintain the testicle in the scrotum, even when it was found to be there, as it would slip back with the greatest facility. Dr. Lovett finally succeeded in seeing the boy at a time when both the testicle and the hernia were down, and in reducing the hernia while the testicle was kept in the scrotum. A carefully adapted truss was then applied so as to prevent the testicle from returning to the abdominal cavity and the hernia from entering the scrotum.

Tumors of the Testis.—We may at birth find an enlargement of the testis due to sarcoma, teratoma, or carcinoma. The former is much the more common.

Malformations about the Rectum.—At an early stage of development of the embryo the intestinal canal ends blindly, and afterwards by an invagination of the outside wall a communication is brought about and the stomodæum formed. An analogous process of development goes on at the other end of the intestinal tube, and results in the formation of the rectum and anus. The hind-gut at first ends blindly, then as it descends it is met by an ascending dimple, and usually these two fuse and the *protodæum* is formed.

As in the mouth a series of malformations may arise from a failure in the completion of this process, so in the anal region we may meet with a similar series. The rectum may have come into its normal relations and the anal depression have failed to form, or it may have gone the whole of the distance between the end of the intestine and the skin and yet the final step, the fusion of the membranes, have failed to take place. To both of these, and to any intermediate condition, the name of *imperforate rectum* is given. On the other hand, with the rectum and the anus fused we may, nevertheless, find a thin parchment-like membrane spread over the external orifice just where the skin and the mucous membrane join. This is called *imperforate anus.*

TREATMENT.—When an infant is born the physician should carefully examine it, in order to determine whether it has any malformation. The most important malformations which it is necessary to recognize are those at the anus. Unless an infant has a passage of meconium soon after its birth, an examination should be made in the rectum with the finger, and if the anal opening is found to be closed, either just at the outlet or higher up, we must consider what is to be done to relieve this condition. If nothing but a web obstructs the anus, we can easily break it through with a director and then dilate the orifice with the finger. If there is more than the thinnest bulging membrane, a cutting operation will have to be done, and perhaps a severe one, so that the case should at once be placed in the hands of a surgeon.

Occlusion of the Vagina.—Sometimes we find a thin gray velum extending across the mouth of the vagina from just below the urethral opening to the posterior commissure and blocking up the vagina. It may be complete or partial.

TREATMENT.—This condition should be dealt with while the infant is still young, as, if left until puberty, it will cause a retention of the menses, and, moreover, by that time will have become much thicker and perhaps quite vascular. It is easily broken through in the young child, and if a piece of carbolized cotton be put between the torn edges to prevent them from adhering, the malformation can be cured permanently. Atresia from inflammation of the labia is said to occur in rare instances.

Hypospadias.—The malformation known as *hypospadias* is the result of an arrest of development in the formation of the urethra and of the corpus spongiosum. The urethral groove should normally be converted into a canal by the growth and joining together of its sides. This process begins at the base and extends to the end of the penis. By an interruption of this process the urethra may be brought to an end and open at any point between the peno-scrotal angle and the base of the glans. In the most common forms of hypospadias the glans alone is imperforate.

TREATMENT.—The treatment is wholly by plastic operation, and it requires the most delicate surgery to obtain a good result in the face of the many serious obstacles that this malformation presents.

Epispadias.—The malformation of *epispadias*, in which the urethral canal opens upon the dorsum of the penis, is still more difficult to deal with than is hypospadias. It is commonly associated with extroversion of the bladder, and is very rare.

TREATMENT.—A partial plastic operation and the wearing of some form of urinal constitute about all that can be done for these cases.

Congenital Obliteration of the Bile-Ducts.—One of the rarer forms of congenital malformations in new-born infants is represented by the obliteration of the bile-ducts. The most extended work which has appeared in the literature of this subject is that of Dr. John Thompson, of Edinburgh.

PATHOLOGY.—There are a number of different morbid processes which have been supposed to produce this pathological lesion of the ducts. Each of these processes has in certain cases, in all probability, had much to do with causing the disease, but it is usually the combination of one or more of them which must be considered in determining its etiology. Thus, the results of intra-uterine peritonitis, by compressing the ducts, or by being a source of inflammation which has spread to the walls of the ducts, may finally cause their obliteration. A primary inflammation or lesion of the ducts themselves may produce this result, or it may arise from an actual arrest or defect of development. In this connection congenital syphilis should be referred to as in some cases producing lesions of the ducts, but this and other causes do not necessarily play an important part in the disease.

The complete discussion of the causes of congenital malformation of the bile-ducts would hardly have a place in a general work on clinical medicine, but it is sufficient to say that in the great majority of cases the evidence is in favor of defective development as being the chief cause. This malformation probably affects to a considerable extent the walls of the ducts, and, as Thompson has stated, it consists in the narrowing of their lumen. The interference which is thus caused to the outflow of bile gives rise to a catarrhal condition which finally blocks and obliterates the ducts, owing to the inflammatory process spreading to the walls of the ducts and the gall-bladder. This progressive inflammation goes on slowly

Vhen the lumen of the duct has become so narrowed that the bile not pass freely into the intestine, a cirrhotic condition begins in the es of the liver, and as it progresses interferes with the functions of organ.

t the post-mortem examinations of these cases the liver usually is l to be much enlarged and its tissues to be increased in consistency; of a dark-brown color, owing to the presence of numerous masses spissated bile in the smaller bile-ducts. In a large number of cases is found a complete obliteration of some part or parts of the hepatic, non, or cystic ducts, or of the gall-bladder, while, with very few ex- ons, implication of the blood-vessels is conspicuously absent.

n speaking of the explanation which may be given for the occurrence e symptoms just mentioned, Thompson remarks that the reappear- of the disease in several members of the same family can be ex- ed only by the theory that a congenital defect of development is in cases the cause of the malformation. The fact that the onset of aundice is not contemporaneous with the blocking of the bile-ducts, usually begins several days after birth, he explains as the effect on epatic cells produced by the great changes in the hepatic circulation n occur in new-born infants. The presence of colored meconium in cases and of only white discharges in others is due to the blocking e ducts having occurred at different periods of intra-uterine life.

Vhen in combination with the colorless fæcal discharges green rial is passed during the progress of the disease, this occurrence is bly due to the chemical action on the contents of the intestine, pro- l in various ways, one of which may arise if mercury has been nistered. The tendency to spontaneous hemorrhages may be due to ccurrence of a condition of chronic blood-poisoning, since the arrest e outflow of bile damages the liver to such an extent that its func- are interfered with and organic fluids of a poisonous nature may pass into the circulation. The enlargement of the spleen, the conwhich the secondary changes in the tissues of the liver are causing with the more important functions of that organ.

SYMPTOMS.—The infants who are born with this disease are either icteric at first or become so within the first few weeks of life. They often appear otherwise healthy and well nourished. In some cases there is a discharge of normal meconium followed by colorless dejections. In other cases the fæcal movements are clay-colored from the very first and remain so. The urine is deeply stained with bile. The jaundice is of a dark-greenish tinge, lasting until death. Spontaneous hemorrhage from the umbilical cord commonly occurs within the first two weeks, and in other localities in those infants who survive this early period. The liver and spleen are increased in size. If the infants survive for some months they become more or less emaciated. Convulsions and vomiting are apt to occur, and death usually takes place from exhaustion or from some trifling intercurrent disease.

TREATMENT.—The treatment must necessarily be symptomatic, there being no known means by which we can counteract the results of this malformation.

Congenital Obliteration of the Intestine.—I shall merely refer to a malformation which is represented by an obliteration of the intestine. Malformations of this kind may arise from constrictions of the parts affected by fibrous bands, probably the remains of peritoneal adhesions.

Congenital Malformations of the Œsophagus and Stomach.—Congenital malformations of the œsophagus and stomach are rare, and are described, in connection with diseases of these parts, on page 274.

Malformations of the Heart and the Blood-Vessels.—The various anomalies of the heart and blood-vessels are described on page 720.

DISEASES OF THE EXTREMITIES.

Fingers.—Various malformations of the extremities are met with in new-born infants, one of these is six instead of five fingers.

Another malformation of this kind, called webbed fingers, is quite common. Surgical interference is indicated in both cases.

Toes.—Infants are at times born with *extra toes* and *webbed toes*, and it becomes a surgical question to determine whether they shall be operated upon. This, of course, is a question of orthopædics, and is one which we need not deal with except so far as to appreciate the importance of preparing the foot properly for future use. The greater freedom of movement required for the fingers, and the fact that the hand is always in sight, render surgical interference much more necessary in malformations of the hand than in those of the foot.

Congenital hypertrophy of the feet and hands, and *congenital deficiency* of one or more extremities, may be spoken of in this connection, but are too rare to be more than referred to. These malformations have been thoroughly described by Thomas Annandale.

Club-Hand and Club-Foot.—Club-hand and club-foot are congenital malformations which may be due to an undeveloped condition of either the bones, the ligaments, or the muscles. In the more simple forms the extremity is pulled into the malposition by the action of contracted muscles and tendons, while in the severe forms the bony framework may be so misshapen that the separate segments are almost unrecognizable. Club-hand is often accompanied by absence of the radius.

TREATMENT.—The treatment of this class of deformities is, of course, purely in the province of the orthopædic surgeon. All that I wish to do in referring to them is to suggest how much may be accomplished by simple manipulations with the hand. The mother should be instructed to rub the foot and leg twice daily, and to make firm pressure against the shortened muscles by trying to bring the hand and foot into the normal position. I have seen slight cases cured by this simple means, and even moderately severe ones so much benefited that subsequent treatment with orthopædic apparatus became much easier.

Congenital Dislocation of the Hip.—Congenital dislocations of all the joints are sometimes found, the most frequent and most important being dislocation of the hip. This is now thought to be caused by a faulty development of the acetabulum and the head of the femur.

SYMPTOMS.—The symptoms are of a kind that readily escape notice during infancy, and are first seen when the child should begin to walk. It is then noticed, if he can hold himself on his feet at all, that the abdomen is very prominent, the back arched, and the buttocks seemingly enlarged: at least this is the case if the deformity is bilateral, which is the form usually met with. On examining the joint we find that the trochanter is above Nélaton's line, but it can by traction on the leg be drawn down to its proper place without causing any discomfort to the child. If the deformity is unilateral, one leg will appear shorter than the other, and the child will walk with a rolling limp. This condition should be carefully looked for when an infant at the age of fourteen or fifteen months has made no especial attempt to walk, or when on attempting to do so it does not succeed.

TREATMENT.—As operative treatment has not proved very successful in these cases and is not to be employed until the child is over three years old, the best method of treatment is by massage. If the disease is unilateral it should in addition to the massage be treated with a high shoe.

Congenital Dislocation of the Knee.—Next in order of frequency to congenital dislocation of the hip, but rare in comparison, is a dislocation, or rather a partial dislocation, of the knee. In this condition the tibia is found riding forward upon the condyles of the femur, so that the knee-joint can readily be put into hyperextension and the toes made to point towards the forehead.

Birth Paralysis.—Birth paralysis will be considered in connection with diseases of the nervous system. It may be present either in the muscles of the face or in those of the extremities, and is due to pre upon the nerves made by the forceps or by too great traction.

GENERAL DISEASES.

Asphyxia.—The earliest pathological condition which is brough our notice at birth, and one which requires immediate treatment, is phyxia. This condition, which is a failure of the circulatory mechan to assume its extra-uterine function of oxygenating the blood, endang the life of the infant from carbonic acid poisoning. It may arise eit from mechanical pressure, as from winding of the cord around the ne from an incomplete expansion of the pulmonary alveoli, *atelectasis,* from other causes connected with the imperfect oxygenation of the blo of which we have very little knowledge. In any case the cause, if know must be quickly removed. This class of cases belongs so directly the province of obstetrics that it need hardly be more than mentione Prompt measures for performing artificial respiration, as by Credé's metho and the stimulation of the pneumogastric nerve by the application of hea cold, and electricity, should be borne in mind: they are well described Dr. Edward Reynolds's work on practical midwifery.

Acute Fatty Degeneration of the New-Born (*Buhl's disease*).—A affection which has been called acute fatty degeneration of the new-bor was described by Buhl in 1861. It is not a disease of common occurrence, and its etiology and pathology have not yet been satisfactorily determined. Runge, of Dorpat, has written more fully on this disease tha any other author, and I am indebted to him for the careful descriptio which he has made of the affection and the literature which he has collected concerning it.

As the anatomical diagnosis can be made only by using the microscope, the disease has probably often been overlooked, and the cause of death ascribed on the one hand to inanition and on the other to such especial forms of hemorrhage in the new-born as omphalorrhagia and melæna. If the numerous causes of hemorrhage from the cord had been more carefully examined anatomically, the disease would probably not have remained so long unknown.

Etiology.—The etiology of acute fatty degeneration of the new-born is very obscure. The disease occurs in animals as well as in human beings, but the investigations made by different observers both on animals and on infants are so varied in their results that we cannot at present consider that we know much about the cause of the disease. It is significant, however, that Buhl in his classic description of the disease states emphatically that the vessels of the cord are not affected, so that if it is due to sepsis the sepsis must have occurred in intra-uterine life through the mouth, the intestinal canal, or the umbilicus, but without producing any change in the umbilical vessels. This can scarcely be considered probable. We know nothing concerning the etiology of this disease, not even whether it is of intra- or extra-uterine origin.

PATHOLOGY.—The pathological conditions which represent the disease consist of a parenchymatous inflammation, followed by a fatty degeneration of the tissues of the heart, liver, and kidneys, and hemorrhages in the various organs. The post-mortem examination of infants dying of this disease, as a rule, shows the following changes. The cadaver is livid and usually icteric. Hemorrhages and œdema are often found in the skin. The umbilicus and the tissues surrounding it are at times stained with blood, but, as a rule, are otherwise normal. The umbilical vessels are in most cases normal. These hemorrhages are especially found in the dura and pia mater, in the pleura and pericardium, and in the connective tissue of the mediastinum: they also occur in the thymus gland, in the peritoneum, in the muscles, and in most of the mucous membranes. The *brain* is found to be soft, usually full of blood, and, if icterus is present, is stained yellow. The *lungs* often show hemorrhagic infarction, and in the bronchi bloody mucus or pure blood. The alveolar epithelium is in a condition of fatty degeneration. The muscles of the *heart* are friable. In the early stages they are rigid and dark red, while in the later stages they become softer and paler. In almost all of them the process of fatty degeneration is found. In recent cases the tissues of the *liver* are blood-red, while in the later stages they are pale and icteric. The liver-cells contain fat-drops and granules of biliary coloring matter. The *spleen* is usually found to be enlarged, and its parenchyma is soft and almost fluid. Hemorrhages may be found in the walls of the *stomach* and *intestine*, and their cavities are often found to be filled with blood. Multiple hemorrhages are found in the parenchyma of the *kidney*. The cortex is swollen in the early stages, is filled with blood, and is pale and yellowish. The epithelium of the convoluted tubules shows marked fatty degeneration, and the canals are often filled with fatty degenerated material. The process of fatty degeneration does not in all cases affect all the organs. In some the changes may be absent or a parenchymatous condition may be present

SYMPTOMS.—The infants who are affected by this disease are usually born in a condition of extreme asphyxia without any apparent cause for it. Attempts at resuscitation are, as a rule, only partially successful, and at times not at all so, many of the cases dying at once. Diarrhœa is commonly present, and is often accompanied by blood from the rectum. There is sometimes vomiting of blood. Often, after the cord has separated, there may be a parenchymatous hemorrhage, which, although small in amount, is at times sufficient to cause death. There is usually a bluish color of the skin, which changes gradually to yellow or a mixture of yellow and blue. Hemorrhages occur frequently in the skin, the conjunctivæ, the mucous membranes of the mouth and nose, and sometimes the outer ear. Icterus may be present in these cases, and at times may become intense. Sometimes œdema occurs, and without any noticeable ri temperature there may be a rapid collapse, followed by death, comr within the first fourteen days of life. These symptoms are not alwa well marked as I have just described them. The external hemorrl may not occur, and the cyanosis, slight at first, may rapidly increas be followed by sudden death. This sometimes happens so quickly we are reminded of the conditions which are met with in cases of by violence.

DIAGNOSIS.—A definite diagnosis cannot be made without a ca microscopic examination. This disease must not be confounded with p phorus or arsenic poisoning, in which the organs undergo similar p ological changes. The history of the case and a chemical examina of the organs will enable you to eliminate these other causes of fatty generation. The differential diagnosis between this disease and case sepsis in which hemorrhages and parenchymatous changes occur is v difficult. When the vessels of the cord are affected, we must in m cases consider the cause to be septic; when the cases occur in groups, is seen at times in hospitals or other places where a number of infa are gathered together, this same cause must be suspected; also wh putrefactive changes have progressed rapidly in the cadaver we should inclined to regard the case as one of septic poisoning, as these chang according to Hecker, do not occur in the specific disease called fat degeneration.

Fatty degeneration at times simulates so closely the appearanc caused by death from suffocation that its presence becomes a questio of great importance from a medico-legal stand-point. The cyanosis, th condition of the lungs, and the ecchymoses, also the absence macroscop cally of other organic changes, can easily suggest suffocation. For thi reason in all cases of death among new-born children where there is suspicion of asphyxia, a careful microscopic examination should be mad of all the organs.

PROGNOSIS.—The prognosis in this disease is very unfavorable; al the cases in which the symptoms are pronounced die. It is possible that the milder forms of the disease can recover, but as yet we do not know enough about this class of cases to state what proportion of them lives.

TREATMENT.—From what has been said concerning this disease it will be readily understood that the treatment is usually unsuccessful. Stimulants should be used and the food carefully regulated.

Infectious Hæmoglobinæmia of the New-born (*infectious hæmoglobinuria; Winkel's disease*).—Infectious hæmoglobinæmia is an affection which is met with in new-born infants usually in the early days of life, and, as a rule, arises as an endemic disease in hospitals. A specific micro-organism has not yet been discovered, yet the endemic character of the malady and the changes which are produced in the blood warrant us in supposing that it is an infectious disease. Although it had been described at an earlier date, yet the most systematic description of it which had appeared up to the year 1879 was that by Winckel, who in that year reported twenty-three cases of an endemic affection observed by him at the Dresden Lying-in Hospital. The disease was characterized by extreme cyanosis, icterus, hæmoglobinuria, somnolence, rapid collapse, and the absence of fever. Although in many respects it resembled closely the acute fatty degeneration described on page 312, yet it had such characteristic symptoms and conditions of its own that for the present it should be kept separate from that disease. I am indebted to Runge for a description of this disease. An analysis of Winckel's cases shows that it usually begins on the fourth day of life, and that it may attack strong, well-developed infants. The course of the affection is very rapid, its average duration being about thirty-two hours. Twenty-five and a half per cent. of all the children born at the time when this epidemic occurred had the disease, and of these nineteen per cent. died.

Etiology.—The etiology of this disease is obscure. Winckel had the organs carefully examined chemically for poisons, such as phosphorus, arsenic, and chlorate of potash, but with negative results. Examinations in regard to carbolic acid poisoning have also been made in these cases, with negative results.

The resemblance of this disease to acute fatty degeneration of the new-born is very striking. Most of the symptoms are common to both diseases. Larger hemorrhages are also not uncommon in this disease, but are not so marked as in acute fatty degeneration. The striking points of difference are the presence of hæmoglobinuria and the fact that large numbers are affected at the same time in infectious hæmoglobinæmia, while these conditions have not been found to occur in acute fatty degeneration. In studying the literature of this disease we find a number of observations by different authors. Dr. W. S. Bigelow describes an epidemic at the Boston Lying-in Hospital in which the chief symptoms were a dark color of the skin resembling somewhat that produced by the administration of nitrate of silver, hæmoglobinuria, diphtheritic deposits on certain of the mucous membranes, and dark brown fæcal dejections. In this epidemic ten infants were attacked and eight died, the average duration of the disease being five days. In one of these cases phlebitis umbilicalis occurred. Similar cases have been reported by Parrot and Herz, in which the urine was brown and strongly tinged with blood, and the kidneys and liver showed the condition of fatty degeneration.

Epstein, of Prague, mentions similar cases in which prominent features were the thickening of the blood, which made it impossible to get a drop to examine, and the dark brown-red color of the urine. Epstein thinks that this disease is a septic process which probably starts in the gastro-enteric tract. He believes that he can controvert the apparent absence of fever by the fact that in the diseases of new-born infants great and sudden variations of temperature occur, and in consequence the perature, for its record to be of value, should be taken very o Whether this is so or not, the disease has certain peculiarities, pointin some cases to an apparent relation with sepsis, and in others to a fatty degeneration. The obscurity as to the etiology of the disease been rendered still greater by the incomplete examinations which h been made of this class of cases, with the exception of those by Win and Birch-Hirschfeld.

Pathology.—A careful post-mortem examination of Winckel's ca showed that there was cyanosis of the external and internal orga Except in one instance, no pathological condition of the vessels of cord was described. The cortex of the *kidney* was found to be wi than normal, to be of a brownish color, and to present numerous min hemorrhages. In some places the pyramids were entirely black-red color, and in other places numerous black streaks were found which co verged to the papillæ. This color was caused by the filling of the straig tubules with granules of hæmoglobin. Intact erythrocytes were nev found. The *bladder* was found to contain greenish-brown urine. T *spleen* was strikingly enlarged and hard. Its length was about 7.5 c (3 inches), and its weight 25 grammes (⅝ ounce). It was black-red color, and on section the surface was smooth. Microscopic examinati showed a considerable accumulation of brownish coloring matter, par free and partly in the pulp-cells.

In addition to these appearances in special organs, minute hemo rhages were found in nearly all the organs, but especially in the pleur pericardium, endocardium, mucous membranes of the stomach and sma intestine, and kidney: they were also found in the dura and pia mate and under the capsule of the liver. The lymph-follicles were swollen, especially Peyer's patches and the mesenteric lymph-glands. A microscopic examination showed fatty degeneration of many important organs, especially the liver, and at times of the muscles of the heart. The bacteriological examinations were, as a rule, negative, especially as regards the tissues of the intestine. Clumps of bacteria were found only once in the liver and once in the kidney.

Symptoms.—The first symptoms were generally restlessness and cyanosis, not only of the face but also of the body and extremities, and especially of the back. The color increased progressively until it became a deep blue. To this was added an icteric color, which when death did not occur within twenty-four hours became very marked. The respiration was rapid; the pulse was not especially increased in rate. The rectal temperature never rose higher than 38.1° C. (100.6° F.). The skin generally felt cool. Vomiting and diarrhœa occurred in some cases. The most striking symptom was the appearance of the urine. It had a pale-brownish color, and was passed frequently, and often with considerable straining. An examination showed that the color was due not to bile, but to hæmoglobin. In the sediment were found numerous epithelial cells from the walls of the kidney, granular casts with blood-corpuscles adherent to them, micrococci, masses of detritus, and urate of ammonia. A small quantity of albumin was present. Later in the disease convulsions occurred, followed rapidly by death. It was noticed that if the skin, where the cyanosis was most marked, was scratched and then pressed hard, a tenacious, almost black-brown fluid exuded. An examination of the blood showed a marked increase of leucocytes and numerous granules.

In other cases besides those of Winckel's in which the blood was examined the condition was found to be one of hæmoglobinæmia. The percentage of hæmoglobin was high, and free hæmoglobin was found in the blood-serum, while the erythrocytes were greatly reduced in number, at times amounting to only 1,700,000 or even less.

TREATMENT.—The treatment should be the administration of oxygen and stimulants, and forced feeding by means of a dropper where the infant is too weak to suck.

Hemorrhage in Early Life.—Spontaneous hemorrhage occurring at some period during the early years of life is not uncommon. These hemorrhages may occur either in the skin or from some trifling traumatic lesion, or they may take place in various internal organs, and especially from the mucous membrane of the mouth and the gastro-enteric tract. A definite division of this class of cases has never been thoroughly made, so that the subject has always been somewhat involved in obscurity. The probability is that these spontaneous hemorrhages are simply symptomatic of different specific diseases, and that as our knowledge of these diseases increases we shall find it necessary to make a clear distinction between cases which now are spoken of under one head. The propriety of separating cases of spontaneous hemorrhage which occur in the early days and weeks of life from those which arise later has been shown by Townsend. He has by a series of observations corroborated the now generally accepted opinion that the hemorrhages which occur in the new-born should be separated from those met with in connection with the hæmophilia of a later period of childhood and of adults. He has called this disease the *hemorrhagic disease of the new-born.* The hemorrhages which occur in new-born infants are so general in their distribution, and yet so uniform in their general symptoms, that they can well be classed under this one heading. These hemorrhages occurring in the early weeks of life run a definite course, and end in death or in complete recovery. The self-limited nature of this affection corresponds to what is seen in the acute infectious diseases, and suggests a relationship to them. The hemorrhage may arise from the gastro-enteric tract, from the mouth, the nose, or the umbilicus, also from the skin, and in the latter case may show itself in the form of ecchymoses. Again, it may occur in the form of hemorrhages in the abdominal cavity, the meninges of the brain, the pleura, the lung, and the thymus gland. Townsend has collected fifty cases of this disease, and has tabulated the sources of the hemorrhage, as follows:

Locality.	Cases.
Intestines	20
Stomach	14
Mouth	14
Nose	12
Umbilicus	18
Ecchymosis in skin	21
Scratch of skin	1
Cephalhæmatoma	3
Meninges	4
Abdominal cavity	2
Pleural cavity	1
Lung	1
Thymus gland	1
From the gastro-enteric tract, nose, and umbilicus, accompanied by ecchymosis in the skin	3
From the gastro-enteric tract alone	19
From the umbilicus alone	3
From ecchymosis in the skin alone	6

The mortality in these cases was 62 per cent. The bleeding first showed itself in all but three within the first seven days of life, the exceptions being on the eighth, ninth, and fourteenth days. The hemorrhage in the majority of these cases began on the second or third day, thirteen starting on the second and sixteen on the third day, while only eight began on the fourth and two on the first day. One-half of the fatal cases lasted one day or less, and all the others died within a week, except one case, in which death took place from the effects of the hemorrhage on the eighth day and several days after the bleeding had ceased. The cases that lived recovered within nine days, and two-thirds of them within five days.

The cases of pseudo-menstruation which occur not uncommonly in the early days of life should not be included in the cases which are classed under the heading of hemorrhagic disease. The *hemorrhagic disease* is apparently infectious and is a general and not a local one, and is found more frequently in hospitals than in private practice. This fact is well exemplified by comparing the percentage of hemorrhagic cases which occurred among 7225 infants observed in the Boston Lying-in Hospital and its out-patient department. The percentage of the disease in the hospital itself was represented by 0.57, while 0.10 represented the proportion outside of the hospital. In Townsend's fifty cases the proportion of females to males was as 20 to 30. In four of Townsend's cases the hemorrhage took place in several other places as well as at the base of the cord, but the patients recovered and the cord separated, in one case in two days and in the other three in four days after the cessation of the disease, without a fresh hemorrhage occurring.

In fourteen of these fifty cases the temperature was carefully observed, and in all but two was found to be elevated at first from 38.3° C. (101° F.) to 39.5° C. (103.1° F.), and in one case to 41.1° C. (106° F.). After he cessation of the hemorrhages the temperature was normal, and often ubnormal.

To recapitulate: it would seem that we are warranted in considering e disease as one of a general nature, and infectious, for the following asons. (1) It occurs usually in hospitals. (2) It is self-limited in its urse, and, although a dangerous disease, may be completely recovered om in one or two weeks and never return. (3) The temperature is ised during the continuation of the chief symptoms, and becomes normal subnormal when the hemorrhage has ceased.

Ritter at the Prague Foundling Hospital has also noticed a great pre-nderance of cases occurring in hospital deliveries over those which re met with outside of the hospitals.

In connection with the hemorrhage which occurs in the gastro-enteric ct, the tar-color of the intestinal dejections, arising from the hemor-age taking place high up in the intestine, is noticeable. The resem-nce of the color of the dejections to that of meconium may cause the sease to be overlooked. A slightly pink tinge on the napkin around e dejection is often, however, seen, and where there is a doubt as to hether the stain is from blood or not, it can usually be determined by eans of the microscope. When the corpuscles have become disinte-rated, as at times occurs, the hæmin crystals may be recognized by means f a simple test which I shall speak of later. The post-mortem examina-ion which was made in nine of these cases throws no additional light upon the nature of the affection. The source of the hemorrhage was found, but in no case were there any gross lesions of the mucous membrane or the blood-vessels. In all these cases the infants looked very anæmic. In one case cultures were made by Professor Councilman from the blood, with negative results. We do not know what the cause of this disease is, but it is probable that in the great majority of cases it has an infectious origin.

The following case is especially interesting, as it shows an unusual result of the blood examination.

The infant was three days old, and presented a blanched appearance of the skin, with stains on the napkin around the intestinal discharges. These stains were examined in the following way. A drop of the semi-liquid dejection was mixed with a little glacial acetic acid and a few crystals of common salt on a glass slide and heated to boiling. On drying the preparation and examining it under the microscope, the dark rhombic crystals of hæmin were easily recognized, showing us that we were dealing with a case of hemorrhage taking place probably high up in the intestine. An examination of the blood by Dr. Wentworth gave the following results:

Erythrocytes	6,245,000
Hæmoglobin	125 per cent.

The blood spread very thickly and stained poorly, but the polynuclear leucocytes appeared greatly in excess of the other forms.

The cases which are commonly designated as *melæna neonato* should be classed under this heading of the hemorrhagic disease of new-born.

An interesting case of this disease was seen by me in consultat with Dr. Bush.

A male infant apparently healthy at birth developed on the third day of its ecchymoses on its head, groins, and one foot. There was also hemorrhage from upper part of the intestine on the fifth and sixth day, the dejections being tar-colo from altered blood which simulated meconium. On the fifth day the child develo a marked paralysis of the left side of the face, and to a less degree of the left arm a leg, presumably from a meningeal hemorrhage. On the seventh day of the disease t hemorrhage had apparently ceased, as the paralysis was beginning to disappear. the twelfth day the paralysis of the left arm and leg had improved: there was, ho ever, still some paralysis on the left side of the face, but this did not continue to a great extent, and in the third and fourth weeks decided improvement took place in t child's condition, and there were no longer any evidences of hemorrhage nor any par ysis. The infant improved rapidly during its first year, and became healthy and stron It learned to walk and talk rather later than usual, but at four years of age was i a normal condition both mentally and physically.

I have met with a number of cases in which these hemorrhages occurred which varied greatly as to extent and persistence. The cases i which umbilical hemorrhage was present showed this same tendency t self-limitation, and could be distinguished from those which are classe under hæmophilia. In fact, it is probable that most cases of umbilica hemorrhage are caused by infection and are not especially connected with hæmophilia.

The following case of umbilical hemorrhage in a male illustrates what has been said concerning the desirability of separating the hemorrhages taking place in the early days and weeks of infancy from those which occur later and in childhood.

The parents of the infant were well and strong, and were Russian Poles. They had another child, eighteen months old, which was healthy. The mother had never had any miscarriages, and stated that her parents were healthy, as were also those of the father. The labor was a normal one, the child presenting in the first position, and nothing abnormal was noticed, except that the placental end of the cord continued to bleed quite freely notwithstanding the application of two ligatures. On the day following the delivery the mother and infant were both doing well; the latter showed slight signs of icterus, but nursed well, and the former had plenty of good breast-milk. The infant continued to thrive, except that there was a slight hemorrhage around the insertion of the cord, which fell off on the eighth day. After the separation of the cord a slight hemorrhage from the umbilicus continued. On the thirteenth day the hemorrhage increased, and became very extensive. The infant was then found to be decidedly jaundiced, though not deeply so. It was nursing well, but looked thin and puny. Nothing abnormal was discovered in the physical examination. Pale watery-looking blood was oozing from the umbilicus, and quite a large cloth had been soaked with the blood, giving evidence of a considerable hemorrhage. The umbilicus was plugged with small pieces of lint soaked in perchloride of iron, firmly compressed by a bandage, and alternate drop doses of fluid extract of ergot and tincture of chloride of iron were ordered to be given three times a day.

On the fifteenth day the hemorrhage had somewhat abated, but it was not thought advisable to remove the bandage; the ergot was omitted, on account of nausea and vomiting. On the sixteenth day the infant was reported to have vomited and cried a great deal, and the plugs of lint had been forced out of the umbilicus, leaving a bleeding surface; the umbilicus was then tamponed with Monsel's solution of subsulphate of iron; the tincture of chloride of iron was omitted, as it caused vomiting. The hemorrhage then lessened and at times ceased. On the seventeenth day the older child pricked the infant's lip with a pin, and on the eighteenth day the lip was found to be still bleeding. The point of hemorrhage was cauterized with a stick of nitrate of silver. This controlled the flow of blood for about two hours, when it returned and continued. Compression of the lip finally stopped the bleeding on the twenty-fourth day. On the nineteenth day the hemorrhage had ceased at the umbilicus, and the child continued to nurse well. On the twenty-ninth day the bandage and lint were removed from the umbilicus, and the abdomen was washed. There was no bleeding; the child looked better, and there was not so much icterus. On the thirtieth day the infant was reported to be perfectly well. It continued to thrive from this time, with no recurrence of the hemorrhage. On the sixtieth day, although I advised that the operation should not be performed, the infant, in accordance with the Jewish custom, was circumcised. I was present at the circumcision, to see if the hemorrhage would be easily arrested. The circumcision was performed without accident, and the hemorrhage was immediately arrested by a weak solution of iron. From this time there was no hemorrhage, and the child continued to be strong and well.

This case is an instance of the self-limitation of the hemorrhagic disease of the new-born, since, although it was a pronounced case of umbilical and general hemorrhage at the beginning of the infant's life, this tendency had ceased by the end of the second month, as was evidenced by the ready control of the hemorrhage after the circumcision.

In another case which came under my notice the hemorrhage took place from the umbilicus in the early days of life at the time of the separation of the cord, and was completely uncontrolled even by ligatures passed around needles introduced through the skin of the abdomen on either side of the umbilicus. This case eventually recovered.

In none of these cases has a tendency to bleeding developed in later life.

At times we meet with what are apparently very mild cases of this disease. The following case was seen in consultation with Dr. George Haven.

The infant, a girl, well developed, and weighing 3358 grammes (about 7 pounds 6 ounces), was born at 12.45 A.M. Nothing abnormal was found on examining it, and it was perfectly quiet until fourteen hours after its birth, when it began to be very restless. This restlessness continued, and the temperature, which at birth was 38.6° C. (101.5° F.) in the rectum, began to rise, until at the end of twenty-four hours it had reached 39.4° C. (103° F.). When it was thirty-six hours old, minute hemorrhagic maculæ were noticed, first on the back of its right hand and arm and then on the right side of its back. A few hours later a number of these maculæ also appeared on the right side of the chest, near the arm. It nursed vigorously, and did not show any signs of weakness, but its respirations were at times quite irregular. From this time no new lesions of the skin appeared, and no hemorrhages from any other locality, the maculæ gradually fading away in ten days. After the first day the temperature fell gradually, and on the fifth day was again normal. Whether there was any loss of weight during the first ten days of life was not known, as it was considered unwise in the infant's precarious condition to weigh it.

The cord separated on the tenth day without hemorrhage, and subs abnormal symptoms arose, and the infant continued to thrive during the w of its lactation.

Sclerema Neonatorum.—Sclerema neonatorum is a dise occurs in the early days of life, and usually among those who in the midst of exceedingly poor hygienic surroundings an weather. It is characterized by a hardening of the skin and t taneous cellular tissue and by a great reduction in the temperat tissues continue to grow cooler and harder until death, whi usually about the ninth day. It is a rare and exceedingly fatal

It should not be looked upon as a local disease of the sk some obscure constitutional affection of the respiratory and c systems, as shown by the shallow respirations and the diminish of the circulation.

A number of cases of this disease have been reported in Eu several in this country, notably by Osler. Some investigators t they have found characteristic changes in the skin. The obser Northrup, however, who published the first report of a typica this kind in America, seem to show that there is no definite lesi skin. Northrup made a careful study of his case, and has plai by sections of the skin compared with normal control specimen histology of the disease does not reveal any change which can b as characteristic. This case embodied every feature of the ty rema of the new-born. The infant was a foundling, born in a damp habitation, and was the weaker of twins. On the fifth life the feet were found to be swollen, and soon began to give tion a feeling of hardness like that of a board. This condi spread upward to the legs, thighs, hips, shoulders, arms, face, a The whole body felt as though it were half frozen. The temp the rectum was under 35° C. (95° F.). The infant died on day.

Symptoms.—Soon after birth, spots of circumscribed hardne on the skin. These spots soon become diffuse, and the disease as it usually does, in the feet or the calves of the legs, passe thighs to the trunk. It may, however, first appear upon the upper extremities, though not commonly. The skin is waxy ar ing, and is hard and cold; the limbs become thick, stiff, and n The infant soon grows weak and somnolent, and refuses to take the breathing becomes rapid and superficial, the voice is v whimpering, and the pulse small and retarded. Towards the e a discharge of bloody serum from the mouth and nose often o death takes place seemingly from inanition.

Treatment.—There is no treatment which has been especiall ful in this disease, but the affection should be recognized at energetically treated with inunctions of hot oil and by massage and stimulants.

Œdema Neonatorum.—Œdema neonatorum is a rare disease, which some authorities describe as distinct from sclerema neonatorum, the chief difference being that the skin pits on pressure and is not so hard as in the latter disease. The general symptoms of the two diseases resemble one another very closely.

Icterus Neonatorum.—Icterus occurs from a number of causes in the new-born infant as symptomatic of disease. There is one form, however, which is of so slight a grade and is characterized by so entire an absence of pathological symptoms that it is usually looked upon as representing a physiological condition occurring in the transition from the intra-uterine to the extra-uterine ciculatory mechanism. It is to this condition that the name icterus neonatorum is given. It occurs in the first few days of life, and may not entirely disappear for several weeks. The most common time for it to begin is from the second to the third day, and, according to its intensity, the usual time of its continuation is from eight to fourteen days. It is not accompanied by any special symptoms. The conjunctivæ are somewhat tinged with yellow in a certain number of cases, but it does not seem to affect the color of the fæcal discharges or to appear in any quantity in the urine.

Careful examinations of the blood in cases of icterus neonatorum fail to show any changes beyond what would be expected in the early transitional stage of blood development commonly found at this age. Plate II., facing page 80, represents a healthy male, ten days old, with the characteristic pigmentation of this disease.

Its weight at birth was 3400 grammes (7½ pounds). At ten days it weighed 3200 grammes (7 pounds). It was perfectly healthy at birth, and its skin was of the usual pink color which is seen in healthy new-born infants. On the fourth day of its life the skin began to show a yellow color, which soon became intensified, representing very well the picture of a physiological icterus neonatorum. The yellowish-brown color of the abdomen, and the slightly icteric color of the conjunctivæ are to be noticed especially. The urine in this case was apparently normal, and the fæcal discharges were tinged with the dark color of the meconium. In a week this yellow color will usually almost entirely disappear, and the skin will assume the natural pink color of a healthy infant in the first month of life. Later it will become whiter and more like the skin of the older child.

As to the cause of icterus neonatorum, the investigations of Birch-Hirschfeld seem to be the most thorough and to offer the most rational explanation. This author says that it is difficult to avoid associating the icterus in some way with a disturbance of the hepatic circulation, owing to the transfer of its chief blood-supply from the umbilical vein. This is especially to be seen when we consider the very evident congestion and œdema of the liver which occur in cases in which the circulation through the umbilical cord is interrupted before the respiratory movements their effect on the right side of the heart, afford an adequate compensat

This explanation of the cause of icterus neonatorum must not, h ever, be regarded as conclusive; for Cohnheim has in a number of au sies made on this class of cases failed to substantiate the conditions scribed by Birch-Hirschfeld.

TREATMENT.—The ordinary bathing of the infant's skin is all tha necessary in these cases of icterus neonatorum, and I have never seen indication for especial treatment of this condition beyond great care establishing the equilibrium between the food and the digestion.

Erythema Neonatorum.—At birth the skin is exceedingly sensit to external influences, and in every case shows variations in color accord to the degree of this sensitiveness, and to the greater or less amount irritation, whether from temperature or from mechanical causes, to wh it is exposed.

The delicate layers of epithelium are commonly thrown off to such degree as almost to represent a physiological desquamation, and it is oft several weeks before the normal infantile condition of the skin is reache In quite a number of cases this natural condition becomes intensified, a we find a uniform redness of the whole skin, which usually appears the first two or three days of life. In a considerable number this hype æmic condition of the skin gradually fades away in about a week, and replaced by a normal pink color. In many other cases, however, the re color may be complicated by an icteric condition, or it may change wit many intermediate shades of red and yellow into a pronounced icteru neonatorum. Plate II., facing page 80, represents a typical case of a uncomplicated erythema neonatorum.

The infant, a female, weighed at birth 3000 grammes (6¾ pounds). It began t turn red when it was twenty hours old, and four hours later was of a dull but pro nounced red color all over its face, head, thorax, and extremities. Its temperatur and respirations were normal, and it seemed perfectly well.

SYMPTOMS.—There are usually no constitutional symptoms in these cases. When the hyperæmia is very intense a slight desquamation is at times noticed. It is well, therefore, to study this rather peculiar red tinge of the skin in comparison with the redness of simple erythema, eczema, erysipelas, and scarlet fever, which, owing to the different degree of sensitiveness of the individual skin, may at times simulate erythema neonatorum as well as each other. The possibility of this error must be accepted, since these other diseases have been known to appear as early in life as in the above case, although it is unusual for them to do so. In one instance, at least, to my knowledge, a case of scarlet fever, which ultimately proved fatal to another child in the family, was considered by the attending physician, when he first saw it, to be a case of erythema neonatorum in the second week of life, and yet eventually he admitted it to be the source of infection of the other members of the family and of the death of one of them.

Treatment.—The application of a simple powder, such as is given below, with the use of water without soap on the skin, using enough only for cleanliness, until the redness has disappeared, is generally all the treatment that is necessary in these cases.

Prescription 51.

Metric.	Gramma.	*Apothecary.*	
℞ Pulv. zinci oxidi	30	℞ Pulv. zinci oxidi	℥i;
Pulv. amyli trit.	120	Pulv. amyli trit.	℥iv.
M.		M.	

Nævus.—There are two forms of pathological disturbance in connection with the blood-vessels of the skin which, appearing at birth, constitute a disease called nævus. Both these forms can appear on the skin of any part of the infant, but its occurrence is especially unfortunate when it is located on the face.

The first form is very superficial in its distribution, and is the one which is usually called "port-wine mark." This form can in a number of cases be destroyed by the use of electricity. The second form, which is deep in its distribution, as a rule needs to be treated by the knife or by the Paquelin cautery. Cases of the superficial form of nævus are quite common and vary greatly in degree. A frequent locality is between the eyes at the bridge of the nose, and another is on one of the eyelids. Often in these cases the disturbance disappears of itself after a few weeks or months and does not return. In other cases the lesion remains, often increases and continues, unless treated, through life. In the second form much can be done by operative interference. This form also varies greatly in size and in the degree of the telangiectasis. In operating on hese cases it should be remembered that at times the hemorrhage is reat, and that the infants are liable to die from exhaustion.

DIVISION VI.

DISEASES OF NUTRITION.

THERE are three diseases which so essentially belong to the early yea of life, and so far as our knowledge of them goes are so obscure in the etiology, while presenting each in its own peculiar manifestations a cle picture of a vice of nutrition, that for the present we must classify them b themselves as dieases of nutrition. These diseases are *rhachitis*, *osteoma lacia*, *infantile scorbutus*, and *infantile atrophy.* Of these, rhachitis an infantile atrophy are diseases of the first two years of life. Infantile scor butus, while having its counterpart in the scorbutus of a later period especially in its pathology and treatment, shows in its symptoms an course such a characteristic picture in the middle period of infancy that i may be considered at least as forming a special form of scorbutus.

RHACHITIS (Rickets).

Rhachitis is a disease closely connected with impaired nutrition. It occurs usually in infancy, rarely in early childhood, and appears at a time when the bones are in the process of development. It is chronic in its course, and although its chief lesions are in the bones, it involves all the tissues and organs of the body. Its chief characteristic is a local or general disturbance of the normal process of ossification, but exactly how the dietetic and hygienic causes which are supposed to produce these osseous changes accomplish their results is still unknown. The uniform and definite changes in the bones in rhachitis lead us to consider it a disease of itself and one which, although connected with nutrition, is not merely a form of malnutrition.

ETIOLOGY AND OCCURRENCE.—There is no evidence that rhachitis is hereditary. Although the disease seldom begins before the sixth month or later than the third year, there is no doubt that it is met with at both an earlier and a later period, and cases have been reported in which the rhachitic rosary was seen as early as the fourth week after birth. The tendency of the disease to occur in the first two years of life is shown in the following collection of 1876 cases compiled by Guerin, Brunische, Rittershain, Ritsche, and Baginsky.

	Cases.	Per Cent.
First year	710	37.8
Second year	831	44.3
Third year	232	12.4
Fourth year	50	2.7
Fifth year	27	1.4
Over five years	26	1.4
	1876	100.0

There does not seem to be any marked difference in the frequency with which boys and girls are affected by rhachitis. Out of 2595 cases reported by various authors, there were 1337 boys and 1258 girls.

Such authors as Ziegler and Vierordt consider that fœtal rhachitis is doubtful, but whatever the pathological point of view may be, a condition closely simulating rhachitis from a clinical stand-point exists occasionally during fœtal life. At about the age of puberty also, in certain individuals, the physical signs of rhachitis, with the exception of a lesser degree of epiphyseal enlargement, occur, and in these cases albuminuria is usually present. These cases are called the *rhachitis of adolescence* or *late rhachitis*. Certain races, such as the lower classes in Italy and England, are notably affected by rhachitis. The disease, on the contrary, in a marked form is not common among native-born Americans, nor is it as a rule, of so severe a type in America as in England; and, except in colored children, or in Italians and Portuguese, great deformities are rare in America. Greeks and Gypsies seem to be almost exempt. The prevalence of rhachitis among the colored population in Northern cities is most striking, and the disease is not so common among the negro population in the Southern States. The great predisposition to rhachitis which has been shown by the people in Southern Europe has never been accounted for. The most severe cases occur in cold, moist climates; it is rare in sub-tropical, almost absent in tropical, regions, and almost unknown in the Arctic regions. It is seldom met with in China, Japan, Turkey, and the southern portions of Italy and Spain (Palm). Even in the regions where it is prevalent it is not common in the high altitudes. It is probable that a great number of cases occur very early in life, but are so mild in form that the rhachitic lesions do not become sufficiently marked for recognition until later. It is certainly much more common in its occurrence among all classes of life than was formerly supposed, and this is especially true of the population of large cities and towns.

Out of 400 cases examined at the Infant's Hospital in a children's out-patient clinic by Morse, 80 per cent. showed distinct changes in the bones characteristic of rhachitis. This percentage is, however, undoubtedly too low, as besides the fact that large numbers of cases of rhachitis do not present marked deformities of the head or extremities, and are, therefore, not always recognized, it was the case also that many infants only a few days old were included in the 400, and only those cases were called rhachitic in which changes in the bones were evident.

As a congenital disease it is probably associated with lack of prop intra-uterine development, corresponding to the rhachitis which is m with in cases of prolonged lactation. Like all diseases associated wi impairment of nutrition, it is less likely to occur among breast-fed infan than among those who are deprived of their natural food, but if the breas milk is deficient in fat and proteids the disease may develop, and it h been noted in New York (Holt) that among the Italians it is very commo to see marked rhachitis in infants who are getting no food but that fro the breast. In like manner it is more likely to develop in the latter pa of the first year than in the earlier months, since in so many cases th breast-milk deteriorates in quality after the first six or seven months o lactation. The disease also seems to occur when the food is not properl adapted to the especial age, and the later children of large families ar more apt to contract the disease than the earlier ones. According to th investigations of König, Forster, Gomp, and Besanez, it was found tha there was not a deficiency of lime in the food taken by rhachitic children It is most probable, however, that rhachitis is closely connected with something which interferes with the assimilation of the lime-salts. In regard to the especial elements of the food which hold a prominent place in the production of the disease, it seems as though a low percentage of fat, especially when combined with a low percentage of proteids, was conducive to the production of rhachitis. It is therefore very evident how the proprietary foods become a prominent factor in the production of the disease, for all of them contain a notably small amount of fat. Next to the food, the general hygiene is of the greatest importance, and the disease is especially noticeable in the crowded tenement districts of large cities. The actual cause and nature of rhachitis is unknown. The disease is not hereditary, but is most apt to develop in children who have inherited a weak constitution.

Rhachitis is a general disease, and there seems to be some change in the metabolism, possibly from the circulation of some substance, in consequence of which there are local changes in the metabolism with a lessened deposition of lime combined with a certain amount of resorption of bone. Whatever the other causes of rhachitis may be, it is evident that interference with nutrition by improper food and lack of suitable hygienic surroundings play a prominent rôle in its production.

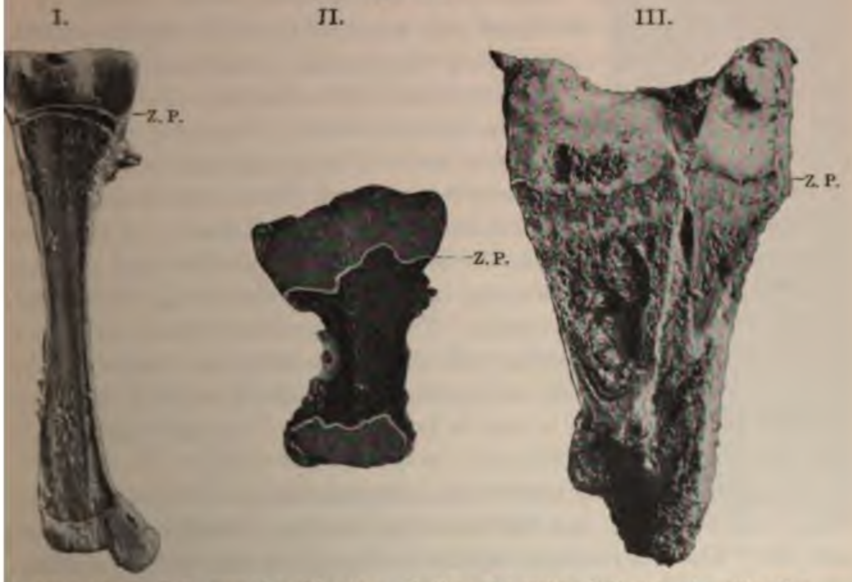
An infectious cause for the disease, though still to be considered, has as yet in no way been proved. The theories of a lack of lime in the circulation, or the circulation of an acid, or a diminished alkalinity of the blood can be eliminated as causes. The growth of cartilage and of osteoid tissue may be the result of lack of lime, or may perhaps be primarily produced and may be an agent of bone change, but there is no proof of this. The vascular inflammatory theory of Kassowitz is not probable, and Oppenheimer's theory of malaria is without foundation.

In addition to the causes of rhachitis, in which the etiology is some-t obscure, many cases occur which evidently are secondary to and lt from a number of diseases which affect the general nutrition of infant. Thus, diseases of the gastro-enteric tract, when they are onged for some time, lead to the condition of rhachitis. We also t with it in cases of syphilis in which the nutrition is much reduced; beyond this relation between syphilis and rhachitis,—namely, that chitis is secondary to syphilis, as it might be to any wasting disease, two diseases are essentially distinct. It is to be noted in this contion that cases of infantile atrophy, no matter how much they are ted, do not present the lesions of rhachitis, although in some cases we the two conditions associated. We must also remember that the rition of all the tissues is profoundly affected, and that the equilibrium he nervous system in rhachitis is very unstable.

PATHOLOGY.—*Bone Lesions.*—The pathological lesions of rhachitis are resented chiefly in the bones, and occur during the period in which normal processes of ossification are most active,—that is, during the year and the first part of the second year of life.

The normal growth of bone depends upon four conditions: (*a*) multiation of cartilaginous cells in definite lines, followed by (*b*) calcifican of the intercellular spaces; (*c*) the formation of medullary spaces by penetration of blood-vessels, with subsequent absorption of tissue; d, (*d*) finally, the concentric deposition of bone within the medullary ces. The bones grow in length by the production of bone-tissue in cartilage between the epiphysis and the diaphysis, and in thickness by

FIG. 82.



I. Normal bone: Z. P., zone of proliferation. II. Bone of a cretin: Z. P., zone of proliferation. III. Rhachitic bone: Z. P., zone of proliferation.

As a congenital disease it is probably associated with lack of pro intra-uterine development, corresponding to the rhachitis which is m with in cases of prolonged lactation. Like all diseases associated wi impairment of nutrition, it is less likely to occur among breast-fed infan than among those who are deprived of their natural food, but if the breas milk is deficient in fat and proteids the disease may develop, and it h been noted in New York (Holt) that among the Italians it is very commo to see marked rhachitis in infants who are getting no food but that fro the breast. In like manner it is more likely to develop in the latter pa of the first year than in the earlier months, since in so many cases th breast-milk deteriorates in quality after the first six or seven months o lactation. The disease also seems to occur when the food is not properl adapted to the especial age, and the later children of large families ar more apt to contract the disease than the earlier ones. According to th investigations of König, Forster, Gomp, and Besanez, it was found tha there was not a deficiency of lime in the food taken by rhachitic children It is most probable, however, that rhachitis is closely connected with something which interferes with the assimilation of the lime-salts. In regard to the especial elements of the food which hold a prominent place in the production of the disease, it seems as though a low percentage of fat, especially when combined with a low percentage of proteids, was conducive to the production of rhachitis. It is therefore very evident how the proprietary foods become a prominent factor in the production of the disease, for all of them contain a notably small amount of fat. Next to the food, the general hygiene is of the greatest importance, and the disease is especially noticeable in the crowded tenement districts of large cities. The actual cause and nature of rhachitis is unknown. The disease is not hereditary, but is most apt to develop in children who have inherited a weak constitution.

Rhachitis is a general disease, and there seems to be some change in the metabolism, possibly from the circulation of some substance, in consequence of which there are local changes in the metabolism with a lessened deposition of lime combined with a certain amount of resorption of bone. Whatever the other causes of rhachitis may be, it is evident that interference with nutrition by improper food and lack of suitable hygienic surroundings play a prominent rôle in its production.

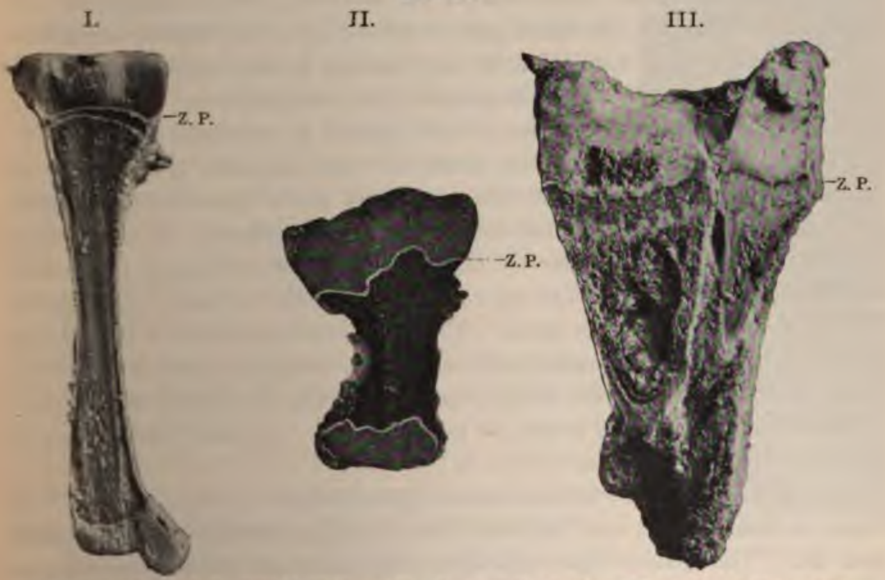
An infectious cause for the disease, though still to be considered, has as yet in no way been proved. The theories of a lack of lime in the circulation, or the circulation of an acid, or a diminished alkalinity of the blood can be eliminated as causes. The growth of cartilage and of osteoid tissue may be the result of lack of lime, or may perhaps be primarily produced and may be an agent of bone change, but there is no proof of this. The vascular inflammatory theory of Kassowitz is not probable, and Oppenheimer's theory of malaria is without foundation.

In addition to the causes of rhachitis, in which the etiology is somewhat obscure, many cases occur which evidently are secondary to and result from a number of diseases which affect the general nutrition of the infant. Thus, diseases of the gastro-enteric tract, when they are prolonged for some time, lead to the condition of rhachitis. We also meet with it in cases of syphilis in which the nutrition is much reduced; but beyond this relation between syphilis and rhachitis,—namely, that rhachitis is secondary to syphilis, as it might be to any wasting disease, the two diseases are essentially distinct. It is to be noted in this connection that cases of infantile atrophy, no matter how much they are wasted, do not present the lesions of rhachitis, although in some cases we find the two conditions associated. We must also remember that the nutrition of all the tissues is profoundly affected, and that the equilibrium of the nervous system in rhachitis is very unstable.

PATHOLOGY.—*Bone Lesions.*—The pathological lesions of rhachitis are represented chiefly in the bones, and occur during the period in which the normal processes of ossification are most active,—that is, during the first year and the first part of the second year of life.

The normal growth of bone depends upon four conditions: (*a*) multiplication of cartilaginous cells in definite lines, followed by (*b*) calcification of the intercellular spaces; (*c*) the formation of medullary spaces by the penetration of blood-vessels, with subsequent absorption of tissue; and, (*d*) finally, the concentric deposition of bone within the medullary spaces. The bones grow in length by the production of bone-tissue in the cartilage between the epiphysis and the diaphysis, and in thickness by

FIG. 82.



I. Normal bone: Z. P., zone of proliferation. II. Bone of a cretin: Z. P., zone of proliferation. III. Rhachitic bone: Z. P., zone of proliferation.

the growth of bone from the inner layers of the periosteum. At same time the medullary canal is enlarged in proportion to the growth the bone by the absorption of its inner layer. These processes progr in definite order and in clearly defined zones.

In rhachitis the chief microscopic features are the changes which occ in the zones of growth and the asymmetrical character of the proliferati processes. The cartilaginous and sub-periosteal cell-growth which pr duces ossification goes on with increased rapidity and in an irregul manner both between the epiphysis and the diaphysis and beneath t periosteum. If we examine microscopically the region between the epip ysis and the diaphysis, usually called the zone of proliferation, we fi that the cartilaginous cells are not regularly arranged in rows around definite zone in advance of the ring of ossification, as in normal tissu but that there is an irregular heaping up of cartilaginous cells, sometim in rows, sometimes not, covering an ill-defined irregular area. This zon of proliferation also, instead of being narrow and sharply defined, is quit lacking in uniformity. It presents a broad, reddish-gray appearance, wit marked thickening and hyperæmia. The medullary spaces are muc more vascular than normal, and are so increased in area as to exten into the zone of calcification, and sometimes through it. The deposi of bone-tissue within these spaces is, however, either absent or ver irregular, and is for the most part replaced by a soft, friable substance, consisting of a bone-tissue that is very lacking in lime-salts, with cells of various kinds embedded in a fibrillated ground-substance. This tissue is called "osteoid," and is similar to that formed by osteoblasts.

In the region of ossification (ends of diaphysis and epiphysis) there is microscopically a pronounced increase of blood-vessels and cartilage-cells, with lengthening of cell columns, and disturbance of calcification of the intercellular substance. Calcification, if present, may be isolated in the region of the proliferating cartilaginous cells, or may be altogether absent over considerable areas. The sub-periosteal layer of cells which is normally thin and scarcely noticeable macroscopically, becomes hyperæmic and thickened with an appearance similar to that of spleen-pulp. Beneath this periosteum is also to be found the "osteoid" tissue seen in the zones of proliferation.

The medulla of the bone is more hyperæmic even than normal. Its tissue is rich in cells, and, like the fœtal medulla, contains dilated vessels and fat. The intercellular substance may show mucoid degeneration or even be of fluid consistency. In such a condition it does not seem that lime is dissolved from the bone tissue by the blood, but it is the resorption of such bone *in toto* that is the important factor in the process. Resorption at the age at which rhachitis occurs is normal. Pimmes, especially, believes that resorption in rhachitis is not increased. Muller and Virchow seem to hold the same views, while Kassowitz and Ziegler think it is increased. Clinically, certain extremely rapid cases of softening

show increased resorption (Vierordt). Ordinarily, with a resorp-
t greatly increased, the formation of fresh bone containing but
me results in loss of strength. In the skull, in some places, ab-
n predominates (occiput); in other cases accretion of osteoid tissues
l and parietal eminences). Deficient bone-growth simply deter-
pen fontanelles. In convalescence lime is deposited in the previous
s osteoid tissue, and the result is a thick and heavy bone. In frac-
t this period callus-formation is intense and excessive.

excessive proliferation of cells in the inner
of the periosteum, the irregular calcifica-
hich occurs about them, and the absence of
nity in the elaboration of the structure of
ne, produce an irregular, spongy bone-tissue
of the compact lamellated tissue which is
essary for the uniformity of the structure.
creased cell-growth between the epiphysis
e diaphysis produces the peculiar knobby
gs which are characteristic of rhachitis.
same time the medullary cavity increases
in size, and the inner layers of the bone
e spongy. The result of these processes is
inish the solidity of the bones so that they
resist the strain of the muscles or outside
re. After a time the rhachitic process may
d the bones may assume a more normal
ter. The porous bone-tissue becomes com-
nd even unnaturally dense, so that in later
od the rhachitic bone is unusually hard,
ry, a condition noticed by those who have
rate on these bones.

. 82, I., represents a section of a normal
aken from an infant, and shows the normal
f proliferation (Z. P.) between the epiphysis
e diaphysis.

. 82, III., represents a section of a rhachitic
and shows the broad, irregular, and abnor-
ne of proliferation (Z. P.) just described.

. 83 represents a section of a rhachitic bone,
ting the great enlargement of cartilage at the epiphysis, with the
ar foci of calcification. The diaphysis of the bone shows perios-
ickening to such an extent that it encroaches on the medullary
which is much diminished.

FIG. 83.

Spindle-shaped rhachitic bone.

eral Lesions; Spleen.—The investigations made by Sasuchin
für Kinderhl., March 13, 1900) have shown that in cases of rha-
which come to autopsy the spleen is invariably enlarged, with the

exception of the cases of clearly pronounced general atrophy, in whic the spleen is of normal size and weight or below normal. A secon marked characteristic is the more or less significant thickening of th capsule and the increase in the consistency of the organ. The thir peculiarity of the rhachitic spleen is the anæmia of the organ and th diminution in the number of Malpighian bodies, which in children ar otherwise well developed, but in these conditions are smooth and hardl perceptible. On section, the spleen has a blood-red color, the trabecula are clearly marked by interlacing threads, and on drawing the knife ove the cut surface there remains upon the edge blood and pulp-tissue. Th characteristics described hold true in all cases of rhachitis, and, in general, indicate approximately the intensity of the changes in the bones.

The microscopic appearances are those of an interstitial splenitis. Whether the inflammatory appearances are directly dependent upon the rhachitis or are due to the complicating diseases which caused the death of the patient has not been definitely determined. The uniform appearances in all the cases examined by Sasuchin, irrespective of the cause of death, seem to give weight to the opinion that the lesions are directly connected with the rhachitis.

SYMPTOMS.—**Constitutional.**—The symptoms of rhachitis are those of a slowly developing constitutional disease. The early symptoms are the same as may occur in a number of diseases in which the nutrition is affected. The most common early symptoms are restlessness at night, profuse sweating, especially of the head, when asleep, and constipation alternating at times with diarrhœa. The appetite is impaired and capricious; the infants are fretful, the abdomen becomes prominent, and although they often grow fat they are anæmic and their muscles are soft. The increase in weight depends mostly on an increase in fat, the normal relative proportion between fat and muscle being altered. The infants do not learn to walk as early as they should; their fontanelles do not close at the usual time; dentition is delayed and irregular, and soft areas appear in the cranial bones, especially in the occiput.

At this stage the characteristic tenderness of the body may occur, but many cases never present this symptom. This is usually due to a periosteal tenderness at the insertions of the muscles, and is sometimes confined to the bones. It is manifested only on trifling pressure, while at other times the muscles are tender and the gentlest effort to lift the child may cause him to shriek with pain. It must, however, be remembered that pain in rhachitis may be connected with an early manifestation of scorbutus, which is not an uncommon complication of rhachitis. The so-called paralysis of rhachitis, which is an accompaniment of this stage, and, as a rule, precedes any marked osseous change, is generally brought to notice by the child's inability or disinclination to walk or to stand. At other times it may be more severe and take the form of inability to use the arms as well as the legs. There may be a slight and irregular heightening of the temperature apart from any complications which can be detected.

Convulsions may occur, especially when there is a tendency to craniotabes. In certain cases these symptoms are all so acute that some writers would make them a separate class under the head of acute rhachitis, but the anatomical lesions and the symptoms are the same, except for their greater severity, and would seem to belong clearly enough to the same group as the less pronounced cases. There has also been much discussion as to whether a form of *acute rhachitis* exists apart from the disease scorbutus. But cases presenting the symptoms to be described under the heading of scorbutus should not be considered necessarily as acute forms of rhachitis on account of the severity of the symptoms, but should be classed as scorbutus supervening on rhachitis. The later and more characteristic symptoms of the disease soon appear and are represented chiefly in the osseous system. The facial expression is usually intelligent.

After these general premonitory symptoms have continued for some time the characteristic changes in the osseous system become prominent and are found in those parts of the bones which are in the most active stage of development. In the early days of extra-uterine life the skull undergoes the most marked changes.

Head.—The typical head of rhachitis has a high, square, prow-shaped forehead, with a decided prominence of the lateral parts of the frontal bones (frontal eminences), and sometimes there are also eminences on the parietal bones. (Fig. 86, page 337.) The normal thickness of the bones is increased by means of a large amount of new periosteal soft growth between the periosteum and the bones. The head is somewhat lengthened beyond the normal shape, and is usually larger in circumference than normal. The posterior fontanelle may remain open for a month, and the anterior fontanelle remains widely open, and may not ossify until the third year, or even later. The sutures also remain open longer than normal, and in such cases may result in leaving a depression in the course of the sagittal suture. Sometimes, however, a prominence is found instead of a depression.

The square, lengthened, rhachitic head is shown in Fig. 86, page 337, and in Fig. 87, opposite page 342. Flattening of the back and top of the head and asymmetry of the head may result from softening of the bones. The normal shape is usually regained when the disease is cured. The bones may be soft, porous, and hyperæmic, while at their edges there may be rough bony projections beneath the periosteum.

The name *craniotabes* is applied to an abnormal thinness of portions of the parietal and occipital bones, which are filled only with a fibrous membrane, and which yield to gentle pressure and give a sensation of crackling parchment. Hyperæmia of the brain and meninges, of course, may be an accompaniment of any affection of the skull so severe as this. With this hyperæmia comes the likelihood of hydrocephalus, either external or internal, and the accompanying cerebral changes, so that hydrocephal becomes a complication which is not very rare. This condition of t bone may be only temporary, and the areas of thickening are often a sorbed; but if there is much deposit under the periosteum it will som times remain, and where calcification takes place quickly the thicken areas of the bone will remain unabsorbed throughout life. At times t jaw-bones are affected; the upper jaw is then found to spread behin and to be pointed in front, while the lower jaw is flat in front and bent i at the sides, making an angle at the site of the canine teeth.

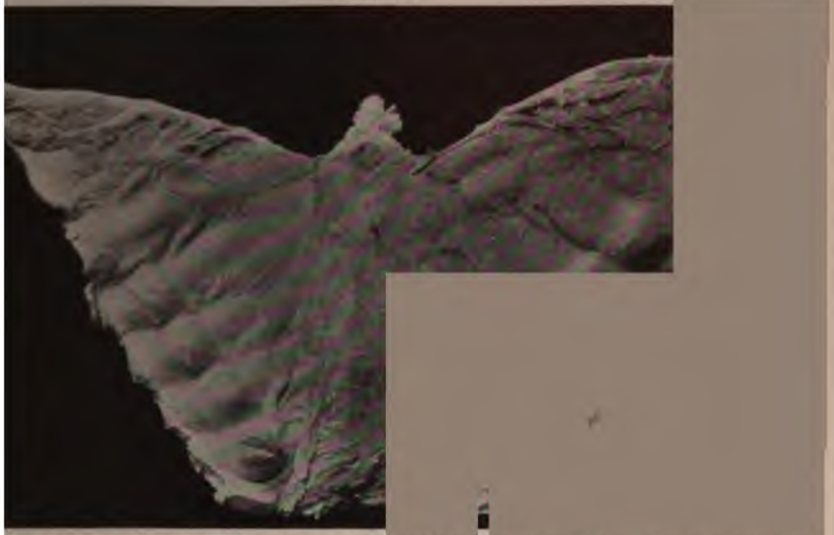
Thorax.—The rhachitic thorax is narrow and is compressed laterall —that is, there is a tendency to a flattening of the sides of the chest an to an increase of the antero-posterior diameter. The forces which pro duce deformities of the thorax are dependent on the muscular action o the soft bones by pulling, atmospheric pressure from without, and th pressure exerted on the bony structures by growing organs.

A transverse depression may also occur, starting at the junction of the ensiform cartilage to the sternum, extending laterally on the thorax, and corresponding to the insertion of the diaphragm. This is called Harrison's groove. The diaphragm, by its strong muscular action along the line of its insertion, may also cause a furrow in the lower part of the chest. The lower ribs may also be elevated by the underlying distention caused by the prominent abdomen and the liver, which is always, even in normal cases, large in proportion to the other organs. Softening of the ribs is said to occur (Vierordt) after the changes in the skull and before the changes in the extremities. In a typical rhachitic thorax the clavicles are shorter and more curved than normal, and the clavicular deformities may be unilateral, as seen in Fig. 87, I. and II., facing page 342. Fractures of the clavicles in rhachitis are not uncommon on the forward curve, and may possibly occur when the infant is being dressed.

When there is unusual lateral compression and narrowing of the thorax, the sternum is made to project forward, and this is called *pigeon-breast*, or *pectus carinatum*. The weakest part of the thorax is at the junction of the cartilages and ribs, and the sternum is thus naturally pushed forward. In another series of cases, in which the ribs are pushed together laterally and the sternum depressed, as where there is interference with the entrance of air into the lungs by adenoid growths and enlarged tonsils, the condition of *funnel chest* is produced, as seen in Fig. 77, page 297. Again, there may be greater compression on one side than on the other, with a resulting prominence or depression on one side of the sternum.

The costal cartilages are frequently enlarged at their junction with the ribs, and can be felt and often seen as a line of rounded prominences. These prominences are called the *rhachitic rosary*, and, though most commonly occurring in the latter part of the first year, have also been met with in the early weeks of life. The rhachitic rosary, according to 's observations on four hundred cases at the Infants' Hospital, is rliest of the physical signs of rhachitis to develop, and is the most

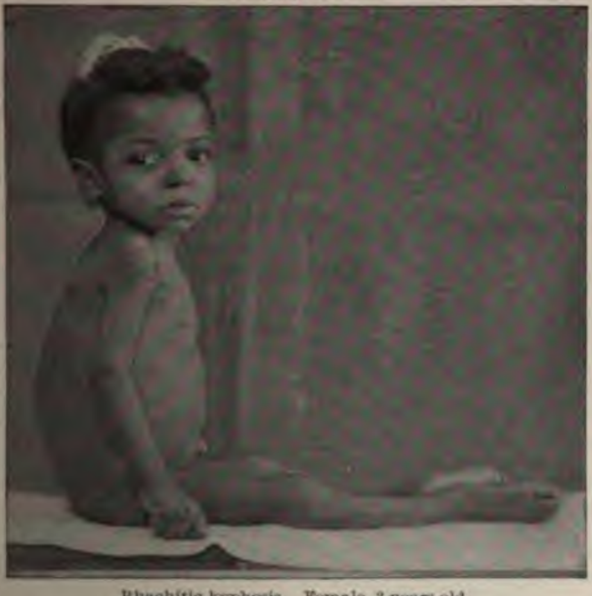
FIG. 84.



surface of sternum, with cartilages and portions of ribs attached, showing rachitic rosary.

on abnormality in rhachitis. It does not occur in normal chil- and its presence justifies the diagnosis of rhachitis. The rosary

FIG. 85.



Rhachitic kyphosis. Female, 3 years old.

netimes present only on the internal surface of the ribs, as seen in 4, which represents a case in which the rhachitic rosary could not be detected on the outer surface. As the pathological process is mo pronounced in the lower ribs, especially the lower five, than the uppe the rosary is more distinct in the lower ribs.

Spine.—Deformity of the spine is quite constant in rhachitis and resul mostly from muscular weakness, but the vertebræ may be affected by th rhachitic process, and in cases of recovery may be found thickened. Th most common deformity is *kyphosis*, which consists of a gradual bowin backward in the dorsal and lumbar regions, as seen in Fig. 85.

The prominence of these vertebral spines is often quite sharp, and simulates Pott's disease, but the rhachitic spine should be flexible to passiv manipulation. In certain cases, however, it is rigid. *Scoliosis* (latera curvature) and *lordosis* (forward curvature) are common deformities occurring in rhachitis.

Extremities.—Deformities of the long bones may arise in the epiphyses and in the shafts: the former show enlargement, the latter bending. Enlargement of the epiphyses appears especially at the wrists and at the anterior ends of the ribs; enlargement of the lower end of the radius and ulna is practically universal, whereas the enlargement of the lower end of the tibia and fibula occurred in four hundred out of a series of one thousand cases. The increase of the epiphyses at the wrists is greater than elsewhere. These enlargements do not involve the joints. In the deep-seated epiphyses, like the hip and the shoulder, the changes are not noticed so readily. The proliferating layer between the epiphyses and the bone may become so thick and softened that consequent deformity and separation of the epiphyses may occur, but such an event is uncommon. When fractures occur they are on the concave side of the bone. In rhachitis of the extremities the curve of the forearm may be due to the pull of the muscles, and there may be a special bend of the radius about the ulna, probably secondary to the antero-posterior curve. It prevents full supination. The humerus rarely bends. Fractures are not rare. In the acute forms of rhachitis the legs rotate outward in bed. Any considerable increase in the ankle epiphysis is rare. The curve of the femur is forward or forward anteriorly, so that in the latter case there may be no curve on the posterior surface. Fractures may occur with excessive callus. In the lower leg fracture takes place most often with anterior bow-legs, and the resulting callus is less than in the thigh. Fractures of the long bones, however, are uncommon, but their arrested development may cause permanent shortening. Bowing of the legs, knock-knee, and flat-foot are all very common symptoms of rhachitis. Coxa vara may be present. Localized rhachitis, as of the legs, is possible and quite frequently met with in children with no symptoms of general rhachitis. It is not unusual to find hydrocephalus combined with rhachitis.

Signs of previous rhachitis are suggested by a caput quadratum with a thick skull, irregularity of the teeth, eversion of the lower edges of the

PLATE V. *A.*

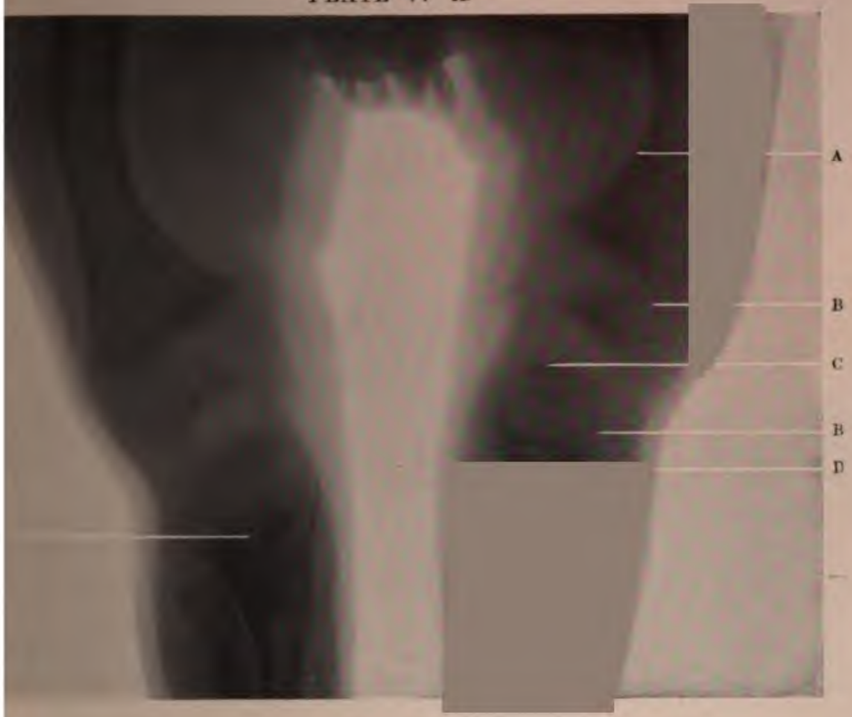
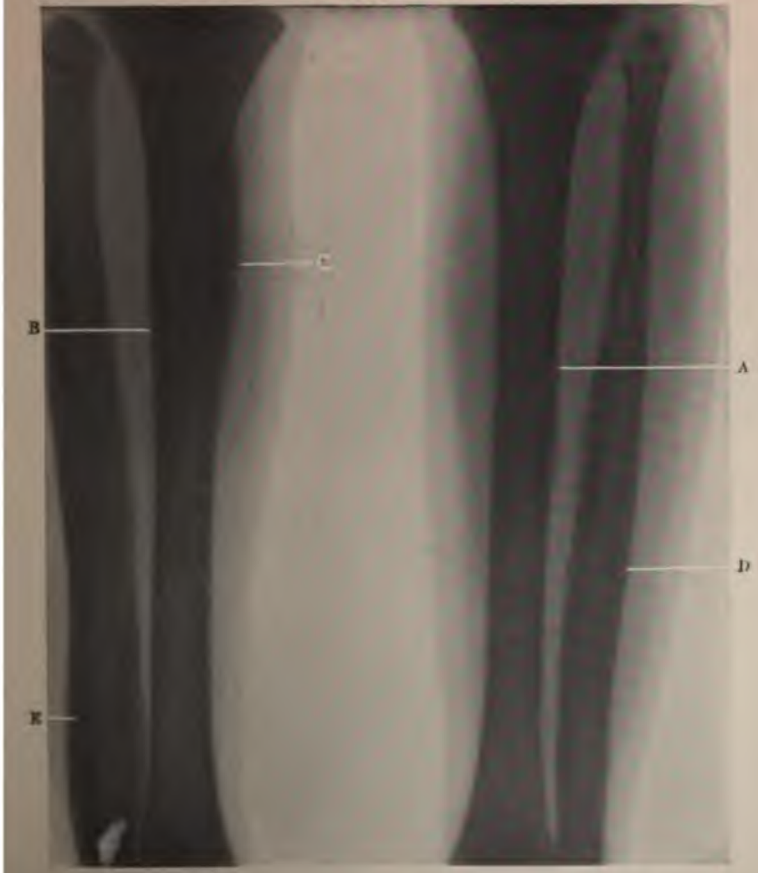


PLATE V *B*



thorax, pigeon-breast, pelvic deformity, deformities of the clavicles and extremities, and thickened epiphyses, which may persist, as thickened epiphyses in general are slow in disappearing and outlast the active process.

The existence of flat-foot in children over two years old should lead to an examination for knock-knee. The combination of these two conditions will in most cases be found to be dependent upon present or previously existing rhachitis.

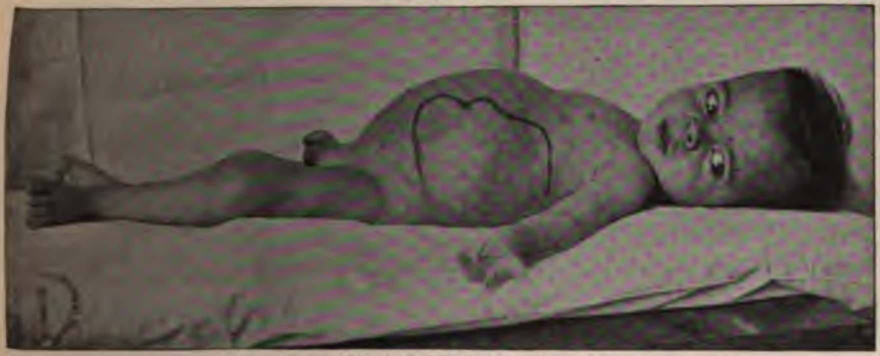
Muscles.—There is a weak and relaxed condition of the muscles, but microscopically the muscles are only pale and their fibres infiltrated with fat, although in some cases there may be atrophy from disuse.

Nervous System.—The nervous system is in an exceedingly unstable and sensitive condition. Convulsions are quite common, especially in connection with craniotabes. The condition of spasm of the glottis (laryngospasmus), although it may occur in other diseases, is especially characteristic of rhachitis. The infants hold their breath, grow cyanotic, are seemingly unconscious, fall back entirely limp, and after a few minutes recover. Tetany at times is met with as a complication.

Lungs.—Owing to deformities of the thoracic walls there is a tendency to atelectasis of portions of the lung from pressure. There is also a great tendency to bronchitis and to broncho-pneumonia.

Heart.—The heart often shows signs of mechanical irritation, represented by irregularity and caused by deformities of the thorax.

FIG. 86.



Male, 3 years old. Rhachitis, with enlarged spleen.

Blood.—Unless rhachitis is accompanied by a secondary anæmia, the blood is practically normal, and presents no other characteristic changes. Hock and Schlesinger found that if the secondary anæmia was moderate in intensity, and diarrhœa and vomiting occurred, it simply made the anæmia more acute. The majority of the leucocytes were found to be mononuclear and about the size of the erythrocytes. There is a moderate permanent leucocytosis in most of these cases, and at times the mononuclear leucocytes seem to be the most numerous form.

Spleen.—In some cases the spleen is found to be enlarged, but it is rarely very large.

The following case, Fig. 86, p. 337, is interesting as an illustration of rhachitis with a secondary anæmia of high grade, accompanied by enlargement of the spleen.

The child was three years old, and was fairly well nourished. It had, however, enlarged epiphyses, a rachitic rosary, the square rhachitic head, and marked bowing of the legs. On physical examination no indication of enlargement of the liver or glands was found. The spleen was very much enlarged, and the position of its outline and its notch is indicated in black. The blood examination showed a severe secondary anæmia, but no evidence of a leucæmia.

Liver.—The liver often shows fatty infiltration, and is at times enlarged but less frequently than the spleen. A distinction must be made in these cases between an enlarged liver and a depressed liver, caused by rhachitic compression of the thorax and narrowing, or from relaxation of the natural ligamentous supports as a result of the weakness from the general inanition which affects all the tissues of the body.

Lymph-nodes.—The lymph-nodes are very frequently found to be enlarged from simple hyperplasia.

Stomach and Intestine.—There is usually, though not necessarily, functional disturbance of digestion; also a subacute catarrhal condition and a tendency to gastric dilatation. The symptoms vary very much and diarrhœa and constipation alternate. Marfan thinks that the intestine is elongated in rhachitis. The distended abdomen results from weakness of the muscles of the abdominal wall and of the intestine, and a resulting umbilical hernia is quite common. In like manner atonic constipation is common.

DIAGNOSIS.—The diagnosis of rhachitis cannot be made by the premonitory symptoms, as the disease is so often the result of impaired nutrition arising from many causes, that it is difficult to determine when the rhachitic symptoms begin. The differential diagnosis has to be made from a number of diseases in which the general nutrition of the child is profoundly disturbed, these diseases being especially represented by functional disorders connected with the gastro-enteric tract. When the pathogenic changes in the bones have progressed sufficiently for physical detection, and the disease is fully developed, the diagnosis is not difficult. In its early stages, therefore, the manifestations of rhachitis may be so slight that the diagnosis must often be held in abeyance.

The differential diagnosis of rhachitis is to be made from scorbutus, rheumatism, osteomalacia, osteomyelitis, syphilis, paralysis of central origin, Pott's disease, and from the disability to use the limbs, due to simple weakness in infants who are not rhachitic.

Scorbutus is eliminated in the diagnosis by the presence of the various osseous lesions which have just been described as symptoms of rhachitis and by the absence of the characteristic features of scorbutus, represented by tenderness and swelling just above the joints, without much fever, sub-periosteal hemorrhages, and stomatitis ulcerosa.

Rheumatism in its articular form would present such marked symptoms of acute tenderness, swelling, and pain in the joints, combined with a heightened temperature, that it could easily be distinguished from the general tenderness of the bones without much fever, together with the subacute or chronic course and the characteristic enlargement of the epiphyses in rhachitis.

Osteomyelitis.—The clinical symptoms of an acute infectious disease, with sudden onset, and represented by severe constitutional disturbance, heightened temperature, pain, localized tenderness, and rapid exhaustion, serve to distinguish osteomyelitis from the slow development and slight amount of fever met with in rhachitis. In osteomyelitis, moreover, various foci of infection will appear in one or more bones with tenderness at these points and a tendency to suppuration which is not met with in rhachitis.

Osteomalacia.—In very rare cases osteomalacia occurs in children, but it can seldom be differentiated from rhachitis during life.

Hereditary Syphilis.—The diagnosis of rhachitis from hereditary syphilis is, as a rule, not difficult. Syphilis and rhachitis have no direct connection with each other, but are both chronic constitutional diseases, and it is possible to have both diseases occur in the same individual. While in rhachitis the enlargement of the long bones is limited to the epiphyses, in syphilis it is not so limited, but involves the ends of the diaphyses. This enlargement is often accompanied by a condition which closely simulates a callus, and there is a distinct tendency to fracture in syphilis rather than to the bending which is common in rhachitis. The notched teeth and the craniotabes may occur in both diseases, while the lesions of the mouth and lips described on page 527, and the lesions of the skin on page 525, are distinctive of syphilis.

Cerebral Paralysis and Poliomyelitis.—In certain cases of rhachitis, especially in the earlier stages, the so-called *paralysis of rhachitis* occurs, and must be distinguished from paralysis of *central* origin, especially from poliomyelitis of the cord. The differential diagnosis must be made by the absence of the symptoms described under cerebral paralysis and poliomyelitis described on pages 950 and 958, and by an examination of the child in a recumbent posture, which in rhachitis will show that the muscular movements are but little impaired, that the apparent inability to move the limbs and the disinclination to walk are caused by tenderness of the bones and muscular weakness, that the electrical reaction is normal, and that the reflexes are not affected. This pseudo-paralysis is certain to pass off if the child lives. Cases of rhachitis which do not walk until late, on account either of muscular weakness or of tenderness, may resemble cases of organic nervous disease with true paralysis. The diagnosis must rest on the presence of the general signs of these nervous diseases.

Pott's Disease.—When rhachitis causes kyphosis of the spine it may simulate Pott's disease very closely. A prominence may be present at the dorso-lumbar junction, which is a frequent seat of the deformity in Pott's disease, and which, involving several vertebræ, may or may not be obliterated when the child lies on its face and is lifted by its feet from the table. The spine is held rigidly in severe cases, just as in Pott's disease, and the deformity may be angular rather than the usual gradual curve. The co-existence of enlarged epiphyses and other rhachitic conditions makes it very probable that the affection is rhachitic; but both diseases may coexist.

In general, the age of the child, under eighteen months, the absence of much pain, and the existence of other signs establish the diagnosis of rhachitis. Rhachitis is, moreover, in children under two, much more common than Pott's disease. In doubtful cases the diagnosis can be made only after several examinations and a period of two or three weeks of recumbency, under which conditions the rhachitic spine becomes somewhat more flexible. In doubtful cases time alone will establish the diagnosis.

Weakness.—In certain infants who are not rhachitic the power of walking is lost for variable periods, and is due to *weakness* following any disease, whether acute or chronic, which for a time may interfere with the infant's vitality. This condition is difficult to distinguish from rhachitis, but must be differentiated by the absence of other symptoms of rhachitis. Delay in learning to walk should lead us to carefully examine for other symptoms of rhachitis, as it is quite often one of the manifestations of this disease. An open fontanelle after the nineteenth or twentieth month suggests rhachitis, and delayed dentition is also significant. If there are no teeth at the ninth or tenth month, the infant should be carefully examined for rhachitis; at one year absence of teeth almost always indicates the disease. Finally, the rhachitic attitude of an infant with well-marked rhachitis is characteristic, and is shown in Fig. 87, facing page 342.

The infant stands with its thighs flexed, knees bent, back arched, shoulders thrown back, prominent abdomen, head rather rigid, legs apart, and varying degrees and combinations of bow-legs and knock-knee with flat-foot. The cause of this attitude, according to Lovett, may in a measure be a persistence of the infantile position which children assume when they are learning to walk.

Children with rhachitis have weak muscles as well as weak bones, and the condition of such a child approaches that of one who stands and walks with the least expenditure of muscular force.

Hydrocephalus.—The diagnosis between the rhachitic head and the hydrocephalic is usually not difficult. The former is irregularly enlarged, flattened on top, square-looking, and has a normally tense or depressed fontanelle. The latter is regularly enlarged and rounded, with a tense and bulging fontanelle.

PROGNOSIS.—The prognosis of rhachitis is favorable, provided no complications arise. When left untreated the disease may, after a decided degree of deformity has occurred, be arrested spontaneously, the pathological process in the bones cease, and the bones harden in their deformed condition (Lovett). In these untreated cases the younger the child the more unfavorable is the prognosis. A spontaneous arrest of the disease may take place in any of its stages, but, as a rule, if the affection is at all pronounced, serious deformities are usually produced. If a hydrocephalic condition, which at times appears in rhachitis, is present to any degree, if there is much diarrhœa, or if the infant is subject to frequent attacks of bronchitis, the prognosis is very unfavorable. Rhachitic children are more liable to die than other children when they are attacked by such diseases as pneumonia or bronchitis. Attacks of the acute exanthemata are of serious import in these cases. Rhachitic children are especially liable to the invasion of the tubercle bacillus. According to Bradford and Lovett, kyphosis disappears under proper treatment. Lateral curvature is permanent when not treated. As a rule, the epiphyseal enlargements diminish with growth, but to a certain degree remain through life. The craniotabes, laryngismus stridulus, bronchitis, diarrhœa, and paralysis gradually in the favorable cases pass away. Although rarely, death may occur in attacks of laryngospasmus and convulsions in rhachitic infants.

When properly treated, the health of rhachitic children improves slowly, and, unless the deformities which have occurred in the bones have advanced too far, more or less complete recovery usually takes place in the third or fourth year. The arrest of the disease at an early stage is important.

TREATMENT.—The treatment of rhachitis is essentially dietetic and hygienic. The infants should be kept in the open air as much as possible, and should live in rooms accessible to sunlight. The food should be adapted to the age, according to the rules given for the feeding of normal infants during the first two years of life. There does not appear to be any drug which produces a specific effect upon the osseous changes which take place in rhachitis. Phosphorus is considered by some observers to be a valuable adjunct to the general dietetic and hygienic treatment, but, according to our experience at the Children's Hospital, it has not proved to be of any especial benefit.

When the anæmia is marked, iron in some form should be given, and at times an increase in the fat in the food seems to be beneficial. Especial attention should be paid to the correction of deformities by exercises and if necessary by surgical interference. Whenever any tenderness or pain is noticed, the infant should at once have orange-juice given to it, as directed in scorbutus (page 347).

Laryngospasmus should be promptly treated by sprinkling the face and chest with cold water and by lightly slapping the back.

Convulsions should be treated with unusual care in a rhachitic child, in whom they usually arise from depression and resulting over-sensitive condition of the nervous system. Stimulants are usually indicated in these cases.

Fig. 87, I. and II., represents unusually well the deformities in the bones which may arise as a result of rhachitis.

The boy was six years old. He was nursed from the breast for two years, and then given a general diet. He began to walk when fourteen months old, and the mother noticed that his legs began to bend at about that time, but could give very little information about the course of the disease. The physical examination showed the heart's impulse to be about on the level with the sixth rib and in the mammillary line. The heart's sounds were clear and loud. Nothing abnormal was detected in the throat or lungs. The abdomen was large, prominent, and tympanitic; the liver was palpable three fingers' breadth below the ribs in the mammillary line; the spleen was not palpable.

Looking at the child from in front, as represented in Fig. 87, I., the head was square with enlarged frontal prominences. There was a decided bowing of the left clavicle with enlarged epiphyses of the wrists and ankles, outward bowing of the femora, and flat-foot with extreme pronation. The side view, as represented in Fig. 87, II., shows the flattened top of the skull, rhachitic rosary, distended abdomen, lordosis, anterior bowing of the tibiæ, and flat-foot.

CONGENITAL OR FŒTAL RHACHITIS.

Fœtal or congenital rhachitis shows a general similarity to the bone changes of rhachitis. There have been a number of cases. Kaufmann lately reports cases of his own and suggests the name chondrodystrophia fœtalis. These cases are by some writers considered not to be rhachitis at all. Some of the cases reported show histological conditions similar to those found in acquired rhachitis. Vierordt, however, is inclined to think that this is hardly evidence enough, as we know that similar changes may be produced experimentally which are not actually rhachitic, and he considers the existence of a fœtal or congenital rhachitis as very doubtful.

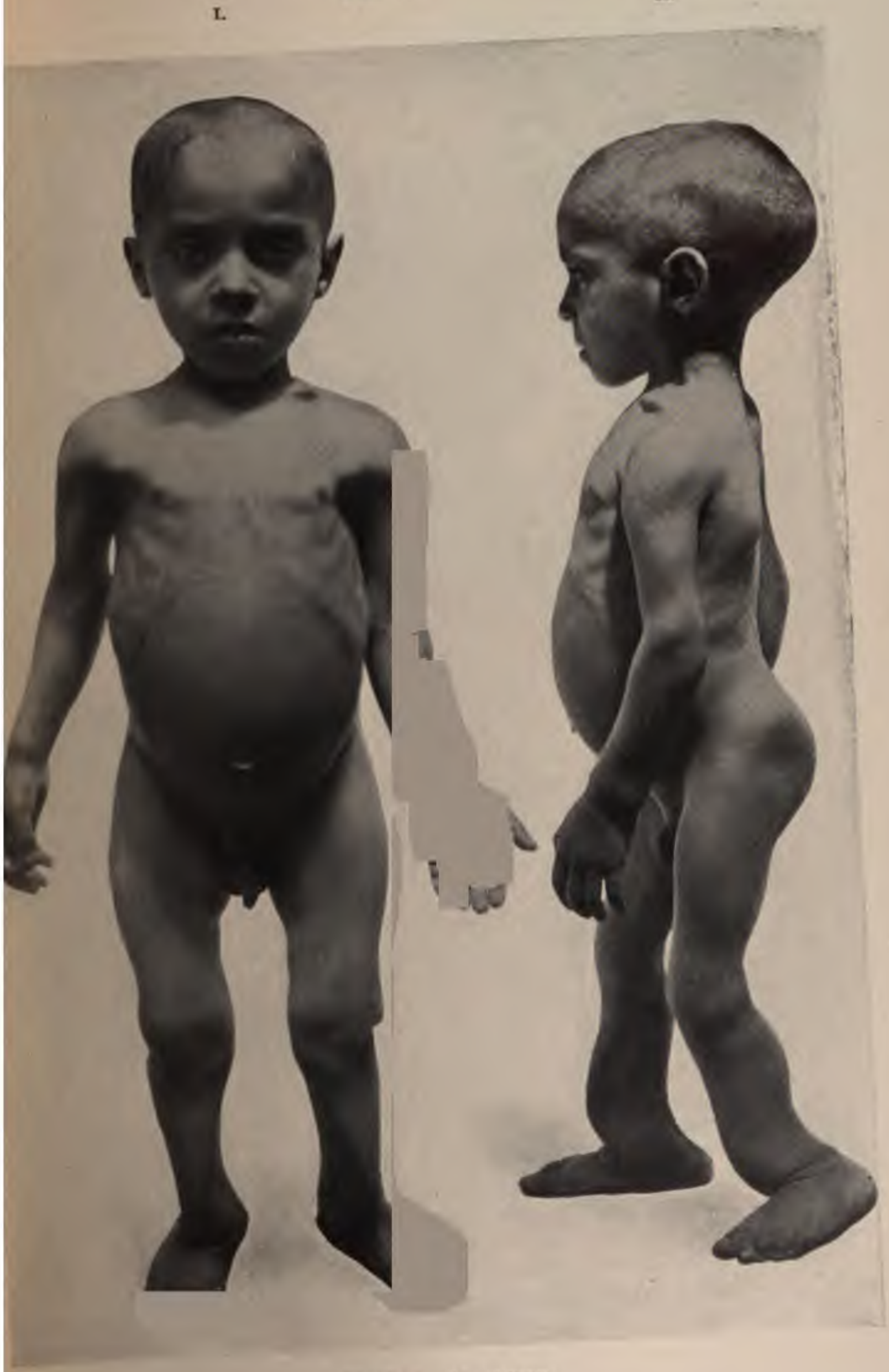
Congenital rhachitis is, therefore, a very rare affection. Ziegler describes various fœtal cases of several prosyllabic types corresponding to Parrot's achondroplasia and Kaufmann's chondrodystrophia.

In these cases of fœtal origin the periosteal changes are absent, and the epiphyseal changes do not correspond accurately to the rhachitis of extra-uterine life. I have seen a case of supposed congenital rhachitis in which the rhachitic process had run its course and the hardening of the bones had apparently been completed before the infant was born.

Another case of congenital rhachitis was seen by me in consultation with Dr. Townsend.

The parents were young and healthy, and there was no history of syphilis or rhachitis. The father was American, the mother Scotch. There was one other child, three years old, strong and well. The mother during her pregnancy was much worried, and her nourishment was both insufficient and poor. The infant, a male, was one month premature. The labor was easy. The infant weighed seven pounds and was 43.3 cm. (17 inches) in length.

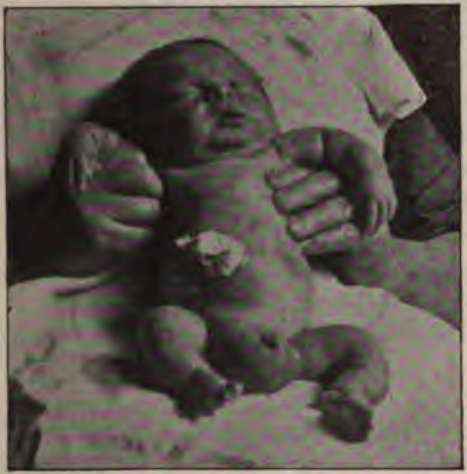
FIG. 87.



Rhachitis. Age, 6 years.

Fig. 88 represents a photograph taken on the fourth day of the infant's life. The head was square in front, was much flattened behind, and measured 33.8 cm. (13¼ inches). The sutures were all widely open. The ossified portions of all the bones of the skull were small, particularly of the occipital bone, which presented a large area of craniotabes. In the widely opened sagittal suture just back of the anterior fontanelle was a large Wormian bone 2.7 cm. (1 inch) long. In the squamous and coronal sutures on the right side at least eight small Wormian bones could be easily felt, and on the left side eleven were counted. The thorax was 30 cm. (11¾ inches) in circumference, and was depressed laterally, the depression increasing with each inspiration, owing to an accompanying atelectasis in the lower portions of the lungs. There was considerable cyanosis. No cardiac murmur was detected. A rhachitic rosary was present. The abdomen measured at the level of the umbilicus 28.7 cm. (11¼ inches). There was a large double inguinal hernia. The spleen could not be detected on examination. The liver could be felt below the edge of the ribs, but was apparently not enlarged. There were marked enlargement of all the epiphyses, curvature of all the long bones, and numerous fractures. The humeri showed a slight anterior curvature. The bones of each forearm were also bent anteriorly. The femora were curved outward and forward. The lower legs showed marked angular curvatures forward at the junction of the middle and lower thirds. The fractures were apparently of as recent origin as the birth, as some of them proceeded to unite very quickly. On the eighth day the fracture of the right tibia was quite firmly united; and only a slight crepitus could still be felt over the left tibia. The fracture of the left humerus was firmly united with a ring of callus. The right humerus at birth showed a callus about the middle of the shaft: this was evidently the repair of an intra-uterine fracture. The child died on the ninth day after birth.

FIG. 88.



Congenital rhachitis.

OSTEOMALACIA.

Osteomalacia is a disease which occasionally occurs in children, but not so frequently as in adults. It causes softening of the bones, and in this respect is somewhat similar to rhachitis.

ETIOLOGY.—Nothing definite is known about the cause, and it is simply spoken of in connection with diseases of nutrition on account of its semblance to rhachitis.

Pathology.—There is, according to Ziegler, an absorption of lime-sal beginning first at the medullary cavity and proceeding outward. T epiphyses are not notably affected by the continuance of the absorptive p cess, the cortical bone becomes spongy and decalcified, and in the sever cases there may remain little but marrow and periosteum (J. C. Warr "Surgical Pathology").

The opinion is generally held that in osteomalacia the layer of ostec tissue results from decalcification, while in rhachitis a similar layer rep sents a new growth deficient in lime-salts. The periosteum is likely be thickened and vascular, and the medulla resembles that in infancy its gross appearance.

Symptoms.—Spontaneous fractures and various distortions may occ in osteomalacia, and the thorax is flattened laterally.

Treatment.—The treatment is the same as in rhachitis (page 341).

SCORBUTUS.

Scorbutus (scurvy) is a constitutional disease closely associated wi imperfect nutrition and having a definite relation to the deprivation the individual of fresh food. It is characterized by anæmia and a te dency to hemorrhage, and in most cases is accompanied by the conditi of the gums which is present in stomatitis ulcerosa.

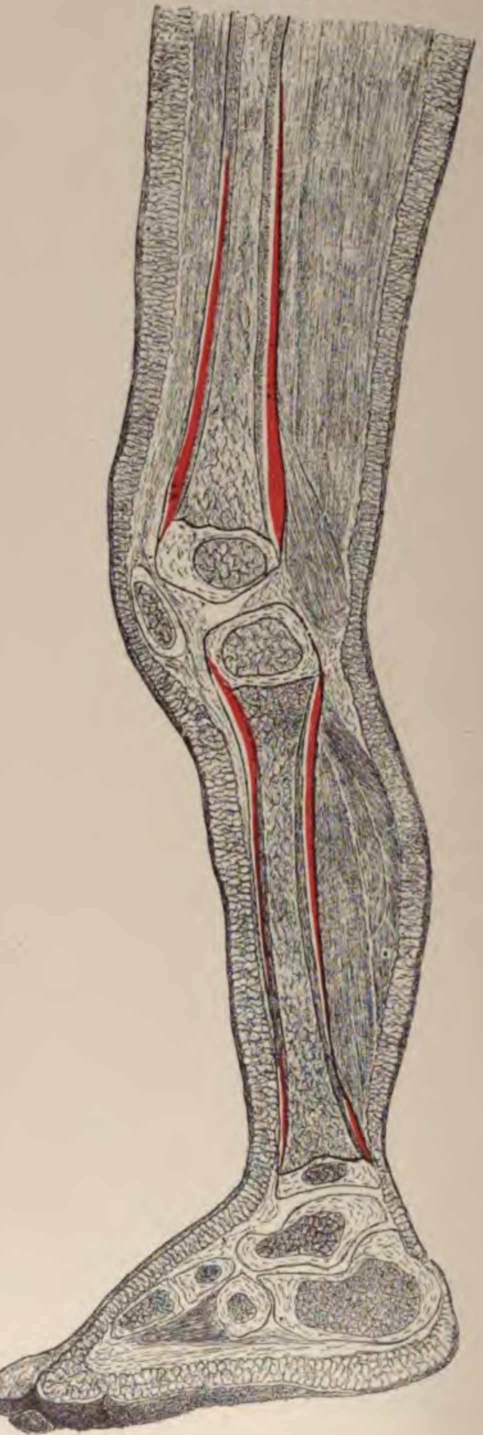
Etiology.—In addition to the view that the cause of scorbutus is chemical origin, owing to the significant relation which the disease has a lack of fresh food, it is supposed that there may be a special mic organism which causes the disease. This, however, has not been prov and we have no further knowledge regarding the etiology of scorbutus

In my experience there is no evidence that sterilized milk is a ca of scorbutus. If the milk is properly modified it can be heated to 7 C. (167° F.), or even to 100° C. (212° F.), without, so far as I am awa having a deleterious effect upon the osseous system.

Pathology.—So few post-mortem examinations have as yet be made on infants dying of scorbutus that the pathological lesions have been finally established. A sufficient number of autopsies, however, been reported by Barlow and others, notably Northrup, to settle at le the more important features in the pathology of infantile scorbutus.

There are no alterations in the blood, either anatomical, chemical, bacteriological, which can be considered peculiar to scorbutus. Th are deep hemorrhages into the muscles and occasionally about or ev into the joints, but the hemorrhage in infantile scorbutus is essentia subperiosteal and chiefly of the long bones. The femora are most co monly affected, and there is a tendency to separation of the epiphys There may also be a varying amount of interstitial hemorrhage in t lungs, spleen, kidney, and intestinal glands. Hæmaturia has been notic

FIG. 89.



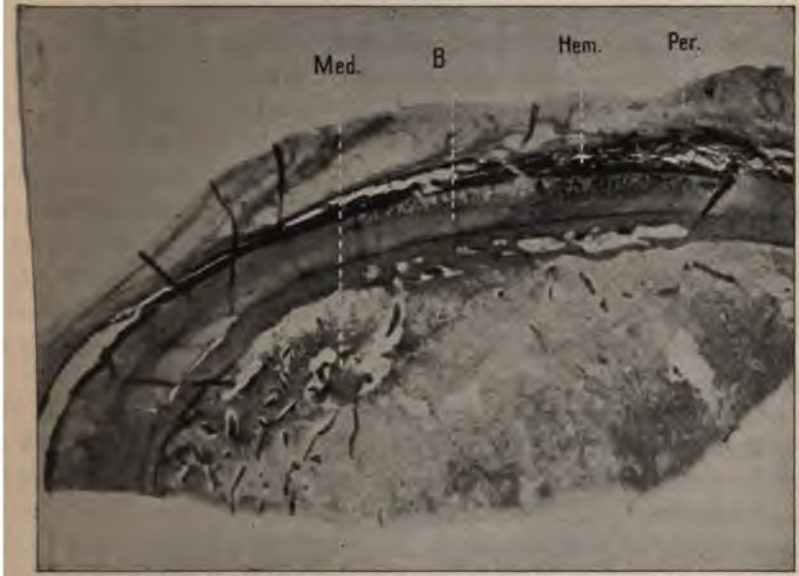
Vertical section of leg in a case of infantile scorbutus. The red areas around the femur and tibia represent subperiosteal hemorrhages. (Specimen preserved in the Museum of the College of Physicians and Surgeons, New York.) (Page 345.)

n a certain number of cases of scorbutus, and it is well to examine the rine in cases in which the disease is suspected. Hemorrhages into the ucous surfaces are usually present, the gums being chiefly affected and esenting the condition of stomatitis ulcerosa.

Fig. 89 represents a section of the bones of the leg in a case of infantile scor-us which was under the care of Dr. Northrup. On examination it will be seen that femur is normal at its upper extremity. The lower half is invested with a black, mous, sub-periosteal layer of blood. The lower epiphysis is detached, and the er end of the shaft, macerated, eroded, and soft, is lying loose in the black, disin-ating blood-clot. The tibia is surrounded by thin, dark, hemorrhagic layers be-h the periosteum, and the proximal portions are congested. The fibulæ and the s of the upper extremities were normal.

Fig. 90 represents a microscopic section of this bone, which shows no syphilitic achitic changes in the bone or the periosteum.

FIG. 90.



Section of scorbutic bone. Med., medulla; B, bone; Hem., hemorrhage; Per., periosteum.

The soft macerated bone gave no evidence of suppuration, but there was a mod-te congestion of the femur and the upper extremity of the tibia.

SYMPTOMS.—The symptoms of infantile scorbutus are those of a slow d progressive cachexia. The infants become anæmic, and show more less gastro-enteric disturbance of a subacute functional type. Profuse eating, especially about the head, at times slight feverish attacks, and sened appetite, are among the early symptoms. The temperature may from time to time slightly raised, but not significantly so. The first mptom, however, which especially attracts the attention is a sensitive ndition of the bones. The infant cries when the affected parts are iched. It does not seem to suffer pain when it is allowed to remain iet, but as the disease advances the expression of its face indicates the fear of being handled. My individual experience with infantile scorbutu has been derived from seventy to eighty cases, all of which, with few exceptions, were from eight to twelve months of age. I met with no case later than the first half of the second year, and with none earlier than th second half of the first year.

As the disease progresses, more marked symptoms develop. Swelling of the limbs, usually of the diaphyses just above the epiphyses, appear These swellings are most common and most prominent in the legs, bu may also appear in the bones of the forearm. They are usually pyriform and symmetrical in shape, the skin over the swelling being more o less tense, but not fluctuating. There is commonly some tenderness on pressure, but, as a rule, no especial heat of the affected part. The pain and swelling do not seem to be in the joint, but in the diaphysis and epiphysis. Signs of hemorrhage may occur in the skin over the affected parts, appearing at first as small blue maculæ and later involving larger areas, as though a deep hemorrhage were coming to the surface. In advanced cases hemorrhage may take place to such an extent in the deeper parts around the eyes that the eyes will be pushed forward (proptosis).

When the infant has not cut any teeth, the mucous membrane of the gums, according to my experience, has not been affected; but when a tooth is pressing on the gum and is almost through, or even when a small portion of a tooth has penetrated the gum, small areas of congested mucous membrane appear, and are of great aid in the diagnosis. In some cases a few hemorrhagic maculæ appear in other parts of the skin, as in that of the forehead.

In addition to the symptoms of epiphyseal pain, the infant keeps the affected limb perfectly still, so that, unless it were understood that it is pain which prevents it from moving the limb, it might be supposed that it was paralysis; in fact, this symptom in scorbutus has been termed pseudo-paralysis. It has nothing to do with true paralysis, and corresponds to what is seen in rheumatic affections of the joints.

DIAGNOSIS.—The diagnosis of infantile scorbutus is to be made from rheumatism, rhachitis, purpura, syphilis, and acute anterior poliomyelitis.

Rheumatism.—In the diagnosis from rheumatism the absence of heat and tenderness of the joint and of a pronounced rise of temperature is usually sufficient to distinguish the two diseases.

Rhachitis.—The diagnosis from rhachitis is to be made by the presence of hemorrhages, the intense pain in the region above the epiphyses, the absence of a rhachitic rosary, and the absence of symptoms of rhachitis when it is not coexistent. If teeth are present, the occurrence of stomatitis ulcerosa usually makes the diagnosis clear. In the cases in which rhachitis is present the symptoms of scorbutus appear to complicate a primary rhachitis, and when the scorbutic symptoms pass away the rhachitic manifestations remain.

Purpura.—Purpura, except in the severe forms in which the joints are affected, is easily differentiated by the absence of the peculiar osseous symptoms of scorbutus.

Syphilis.—Scorbutus is differentiated from syphilis by the extreme tenderness, the hemorrhages, and the commonly occurring stomatitis ulcerosa which occur in the former disease, while syphilis has distinctive symptoms which are not found in scorbutus, which will be described under the former disease.

Acute Anterior Poliomyelitis.—The differential diagnosis between scorbutus and acute anterior poliomyelitis is made by the presence in the former of enlargement and tenderness in the neighborhood of the epiphyses. Pain is present only in the initial stage of acute anterior poliomyelitis, and tenderness is absent. In acute anterior poliomyelitis, also, the onset is sudden, and there are no premonitory symptoms.

PROGNOSIS.—Scorbutus is very variable in its duration. If left untreated, all the symptoms may become more pronounced and the infant finally die of exhaustion. When properly treated, and uncomplicated by any other disease, the prognosis is very favorable if treatment is begun early in the attack, before the vitality of the infant has been too much reduced.

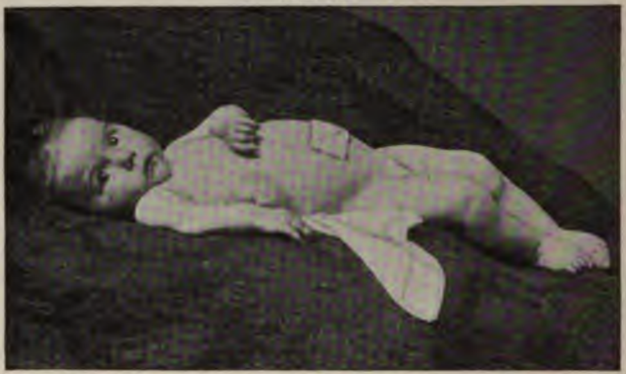
TREATMENT.—The treatment of infantile scorbutus is essentially by changing the improper food which in most cases is being given, to fresh milk and orange-juice. Under this treatment the pain and tenderness of the limbs rapidly disappear, sometimes within a few days, as does also the stomatitis ulcerosa. In the beginning the juice of one orange should be given during the twenty-four hours. It usually is well to dilute the orange-juice one-third with water. If a rapid improvement does not take place, a still larger dose should be given within a few days. These scorbutic infants usually take orange-juice with avidity, but they should be forced to take it if they do not like it. The nurse should be cautioned to move the affected limbs as little as possible, and the infant should be kept on a comfortable pillow on which it can be carried about.

In my earlier cases, before I recognized the scorbutic element in the disease, I treated these infants with a number of drugs, none of which appeared to have the slightest beneficial effect. In some of these cases the symptoms grew progressively worse, and the infants died. In one of them, however, in whom the hemorrhages in the skin were extensive and proptosis was marked, the infant recovered entirely when a properly modified milk was substituted for the artificial food which it had been taking. In some of the later cases which I have seen in consultation, where infants living in the country with good hygienic surroundings were being fed on one of the many artificial foods, the disease had progressed to such an extent that the infants were extremely anæmic, had hemorrhages in various parts of the skin, were unable to take any food, and were seemingly dying; in fact, they were as much reduced as were the cases which I have just spoken of as having terminated fatally. These infants, after taking orange-juice for a few days, invariably improved rapidly, and usually recovered entirely in two or three weeks.

All my cases have presented in different degrees the symptoms which I have just described, and which are well represented in the following case and in Fig. 91:

The infant, a female, ten months old, was healthy at birth and weighed 3636 grammes (8 pounds). It was nursed at first, but later was fed on a patent food, on which it did not gain. When it was eight months old it lost somewhat in weight, had profuse sweating, and began to have tenderness in its limbs. It had six teeth. There was an expression of fear on its face, and it kept its arms and legs perfectly motionless.

FIG. 91.



Infantile scorbutus. (Second month of disease.) Female, 10 months old.

Whenever it thought that its legs or arms were going to be touched it cried with fear. There was no evidence of rhachitis in this infant. There was a swelling of the diaphysis just above the epiphyses of the bones of the right wrist, and also in the lower part of the femur of each leg and the lower part of the tibia. The swelling did not fluctuate, had a hard, tense feeling, and apparently was not connected with the joints. There was no increased heat of the skin, but there were certain circumscribed areas of hemorrhage in the skin over the swellings. The gums showed the condition of stomatitis ulcerosa to so marked a degreé that they almost covered the teeth. They were purple, bled easily, and were very similar to those represented in Plate XI., facing page 620.

The infant's diet was changed to a modified milk, and it was given the juice of one orange daily. Within two days it moved its legs and arms freely, the anxious expression left its face, and in a few weeks it had gained much in weight and was perfectly well.

An examination of the blood in this case showed only a secondary anæmia.

INFANTILE ATROPHY.

Infantile atrophy (marasmus, athrepsia) is essentially a disease of infancy and early childhood, and occurs most frequently in the first six months of life. It is a condition in which extreme atrophy of all the soft tissues takes place without demonstrable disease of any of the organs. It is apparently the result of a vice of absorption, although this has by no means been clearly proved. The wasting which occurs as the result of malformations of the mouth and throat preventing the entrance of sufficient food, or when insufficient food is given, should be considered as the result of starvation. The name infantile atrophy should be restricted to the cases in which such causes do not exist. In like manner the wasting which occurs in the course of constitutional diseases, such as tuberculosis or syphilis, or as the result of severe gastro-enteric disturbance, is not to be considered under the name of infantile atrophy.

ETIOLOGY.—The cause of the primary cases of infantile atrophy is unknown. In a number of cases the disease seems to be secondary to grave intestinal disturbance, whether of toxic or of organic origin. The disease appears to depend upon improper diet or bad hygienic surroundings, either separately or in combination. Thus infants may develop the disease whose food is seemingly good but whose surroundings in crowded cities or districts are unhygienic as to light, warmth, pure air, and air space. On the other hand, the disease is at times met with in the country where the hygiene is good but the food poor.

PATHOLOGY.—The pathological conditions which are found in cases of infantile atrophy are exceedingly unsatisfactory, and have not given us much information concerning the disease. Nothing abnormal is found in the various organs which can be said to be characteristic of this disease. It is supposed by some pathologists that the lymph-glands are enlarged, but this enlargement does not seem to be a prominent feature, and is chiefly confined to the superficial glands of the neck, axillæ, and groins. No pathological condition of the mesenteric lymph-glands has been found, and the atrophy of the mesentery around them is so great that their increase in size may be seeming rather than real. In the intestine, although in some cases there is considerable atrophy of the mucous membrane and the submucous tissue, no characteristic lesion has been proved to be present. Therefore, until our knowledge of the pathology of this disease becomes more definite, it is better for us to consider its pathology as simply atrophy. Certain secondary morbid conditions are frequently found. Prominent among these are anæmia, atelectasis in the lungs, bronchitis, broncho-pneumonia, fatty liver, and intestinal catarrh.

SYMPTOMS.—The essential symptom of infantile atrophy is extreme wasting. An infant which has perhaps been weak and delicate, or may have been seemingly well nourished, begins to emaciate. The prominent feature of the disease is the progressive and extreme loss of weight. The loss of subcutaneous adipose tissue is excessive, the skin is dry and wrinkled, and hangs in folds on the bones, giving the appearance of a living skeleton or advanced old age. The extremities are cool. The abdomen soon becomes sunken. The tongue is dry and usually reddened. The pulse is feeble and usually rapid. The temperature is normal, or more commonly subnormal, but in some cases is raised. The respirations are generally normal. Although secondary anæmia is quite a prominent condition, extreme pallor is not usual. The appetite is usually lessened, but may be at times voracious. In many cases the food is well digested, and the discharges yellow and smooth. In some cases the total amount of the fæcal discharges in twenty-four hours is abnormally large. Regurgitation of food is common, and at times vomiting of a reflex nature becomes prominent. The normal fæcal discharges at times are abnormal in color and consistency. In advanced cases ecchymoses may appear in the skin, sometimes covering the entire thorax.

DIAGNOSIS.—The diagnosis of infantile atrophy is chiefly to be made from ordinary starvation and from general tuberculosis. From the former it is soon differentiated by its lack of response to good food and improved hygiene. In the ordinary cases of starvation which result either from improper food, or from lack of food, a diet carefully adapted to the age of the infant or child is soon followed by rapid improvement. The cases of wasting from mechanical causes are recognized by detecting the special malformation which is present. Those cases which are secondary to other diseases are differentiated from the primary cases of infantile atrophy by the prominence in the history of the essential symptoms of such diseases. In other words, in these cases the wasting is not the one essential symptom.

The differential diagnosis from general tuberculosis is at times exceedingly difficult. I have had under my care in the hospital in adjacent beds an infant with infantile atrophy and one with general tuberculosis. In these two cases the symptoms and courses of the diseases were identical, and it was impossible to differentiate the two diseases except at the autopsy. On physical examination nothing abnormal could be found in either case except extreme emaciation. In both cases the temperature was slightly raised. The physical examination from tuberculosis, therefore, if the temperature is raised, is almost impossible, as complicating processes in the lungs may produce evidence of solidification in infantile atrophy. Where, however, solidification is prominent in the front of the lungs, it is most likely to arise from tuberculosis. The presence or absence of the tuberculin reaction will in many cases be needed to differentiate the two diseases.

PROGNOSIS.—The prognosis of infantile atrophy is bad, especially during the first year of life. Even under the most careful treatment it is always a very intractable disease. Even the most exact modification of the food at times entirely fails to cure the disease. When, however, the infant recovers, the recovery is usually complete, and perhaps within a year the child may show no signs of the previous serious condition.

TREATMENT.—The treatment of infantile atrophy is essentially by such modification of the milk as to promote intestinal absorption, and without drugs. Especial attention should be paid to keeping up the bodily temperature by external warmth and by the administration of stimulants. Small doses of brandy can often be given for weeks with great benefit.

Although, as has already been stated, it is not entirely proved that the morbid condition is that of a lack of absorption, yet my clinical results are most favorable when the disease has been treated on this principle. After experimenting in a large number of cases by modifying the different constituents of the milk in various ways, I have arrived at the following conclusion: a mixture should be given which contains a low percentage of fat, a high percentage of sugar, and a moderate percentage of proteids. The low percentage of fat is given on the supposition that the infant will increase in weight and thrive on a small proportion of fat, provided it is absorbed. I have found that when higher percentages of fat are given the infant continues to lose in weight. The administration of cod-liver oil is not indicated in these cases, for it is only by a precise adjustment of the percentage of the fat in the food to the individual power of absorption that good results can be obtained. The sugar of high percentage and the proteids of normal percentage seem to be digested and absorbed provided they are combined with a low percentage of fat, since by this combination the nutritive properties of the sugar and of the proteids are made use of. The prescription which I usually write in the beginning of the treatment of these cases, when they occur in the first year of life, is the following:

Prescription 52.

Fat	0.50
Sugar	6.00
Proteids (lactalbumin 0.75, caseinogen 0.25)	1.00
Lime-water	5.00

After the infant has begun to gain in weight I usually increase the percentage of the fat, but for a number of weeks I do not raise this percentage above 1 or 2. When the infant has once begun to gain steadily the power of absorbing fat is rapidly regained, and percentages such as are in the following prescription can then be given:

Prescription 53.

Fat	3.00
Sugar	7.00
Proteids	2.00
Lime-water	5.00

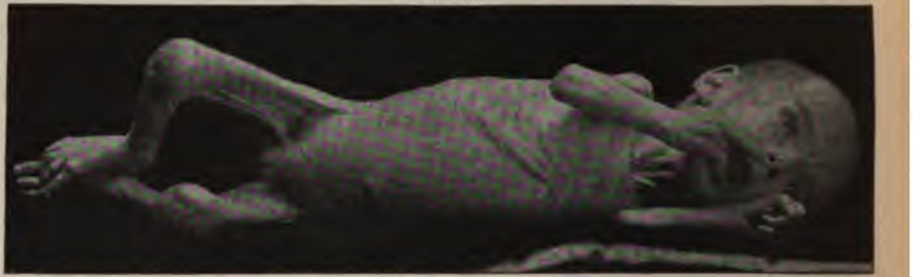
The same treatment can be carried out when the disease occurs in children in their second and third years, but in these cases it is usually possible to increase the percentages of the different elements more rapidly, and after two or three weeks to begin with other articles of diet, such as beef-juice, broths of various kinds, and finally, with caution, cereals.

These special modifications of the milk do not, of course, suit every individual infant or child, and when the treatment with them is not successful, each of the elements of the milk must be carefully changed and different combinations of these elements tried until the individual idiosyncrasy of absorption in the special case has been discovered. A very important part of the treatment is the general hygiene, especially sufficient air space and pure air.

The following cases illustrate infantile atrophy of high grade.

An infant nine months old had been fed on foods of various kinds, all of which contained a considerable percentage of starch. She is said to have been healthy and plump at birth and during the early months of life while she was nursed. After she was weaned and placed on starchy foods she began to lose progressively in weight, and became extremely emaciated. Physical examination showed nothing abnormal. She had four teeth. Her temperature was slightly subnormal, her pulse was regular but weak, her respirations normal. On first entering the hospital the bowels were constipated and the fæcal movements were brown and looked poorly digested. On being placed on a diet of modified milk the movements became well digested and of normal color, but the total amount in twenty-four hours was greater than usual. She was very fretful, and at times vomited, but when her diet was regulated she became less fretful and somewhat apathetic. On entering the hospital she weighed 2966 grammes (6½ pounds). She was in the hospital two weeks, and gained in that time 1000 grammes. The food which was found to suit her powers of absorption contained fat 1, sugar 5, proteids 1, lime-water 5, and 60 to 120 c.c. (2 to 4 ounces) were given every two hours.

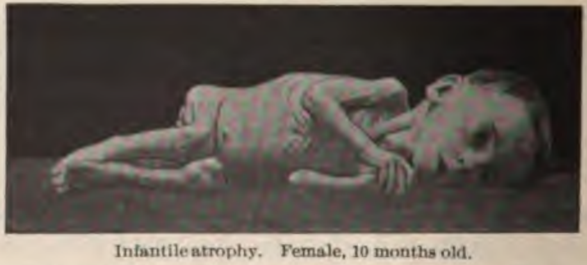
Fig. 92.



Infantile atrophy. Female, 9 months old.

She did not increase progressively in weight, but sometimes lost considerably, and once it seemed as though she could not possibly live. After the food had been modified in various ways, she finally began to improve, and when she was able to digest and absorb 150 c.c. (5 ounces) of milk so modified as to contain fat 3.5, sugar 6.5, and proteids 1.5, she improved rapidly, and eventually recovered entirely. Her temperature, with few exceptions, was normal or subnormal through the whole course of the attack.

Fig. 93.



Infantile atrophy. Female, 10 months old.

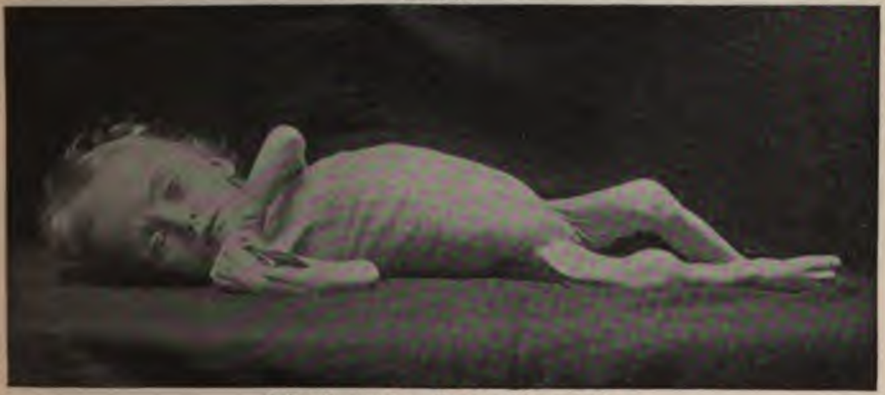
This next case was also one of infantile atrophy of high grade.

An infant 10 months old entered the hospital with a history of having been fed on various foods containing starch from the earliest months of its life. It was said to have been healthy at birth and of average weight. On entering the hospital it weighed 2593 grammes (5¾ pounds). It was extremely emaciated, and illustrated the more advanced stage of infantile atrophy. It was unable to raise its head and was apathetic ; its skin was cool and dry ; its respirations were shallow ; its pulse was weak, and its temperature was slightly subnormal. It looked as though it could not live many days. A physical examination showed nothing abnormal in any of the organs. The fæcal movements were rather large in amount, but when its food was carefully regulated, became fairly well digested. On entering the hospital they were still larger in amount and were of a brownish color. It weighed, after being in the hospital one week, 2570 grammes (about 5½ pounds), which was slightly less than its weight on entrance. It was fed on a modified milk in which the percentage of fat was 2, sugar 6, proteids 1, lime-water 10.

In two weeks the infant began to gain in weight and to absorb its food. Although it had a number of relapses, in which it lost considerably in weight, it finally began to gain steadily. At the end of three months it had recovered entirely, and was quite plump. In this case the percentage of the fat was finally raised to 4, and that of the sugar to 7, but the proteids had to be kept at 1 ; the lime-water was reduced to 5.

The next case was a female, one and a half years old, who, on entering the hospital, weighed 4281 grammes (9½ pounds). She was said to have weighed but 900 grammes (2 pounds) at birth. She was nursed by her mother, who apparently had plenty of good breast-milk, and who had two other children whom she had nursed that were healthy and strong. As the infant did not gain, she was nursed for only a short time, and was then fed on various artificial foods. She began to lose in weight, and this loss continued so that her emaciation was extreme.

FIG. 94.

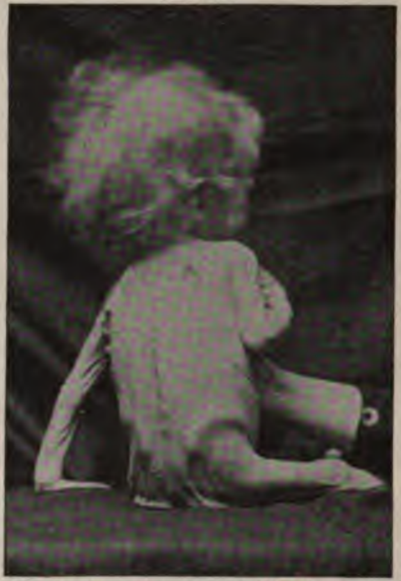


Infantile atrophy. Female, 1½ years old.

On physical examination the anterior fontanelle was found to be widely open. There was no enlargement of the epiphyses of the ankles or wrists, but there was a slight rhachitic rosary. Nothing abnormal could be detected in any of the organs. She had four upper and two lower incisors. She was very apathetic, and seemed hungry, but when food was given to her she vomited. After entering the hospital she lost 519 grammes (1⅛ pounds) in two weeks. Her skin was dry, harsh, and at times quite cold. The fæcal movements were very large in amount, but after entering the hospital were fairly digested. The cervical and inguinal glands were slightly enlarged and she had a slight cough. She lost steadily in weight, did not respond to the various modifications of the milk given to her, and died three weeks after entering the hospital.

The post-mortem examination showed the following condition : There was extreme atrophy of all the muscles. There were no changes in the mesenteric glands, and they were not enlarged, although the extreme atrophy of the mesentery around them made them appear so. The liver was normal and its tissue showed little evidence of atrophy. The spleen was normal. Sections made from various places in the stomach and the intestines showed no changes beyond considerable atrophy of the mucous membrane of the submucous tissues. The thyroid gland was atrophied. There was an extensive bronchitis in the posterior portion of the lungs, while in some parts there was a partial, and in others, a complete atelectasis.

FIG. 95.



Infantile atrophy, showing extreme emaciation of arms, back, and hips.

DIVISION VII.

DISEASES OF THE SKIN.

THE rule that the child should be inspected in every part is very important, and is especially applicable to cases in which there is disease of the skin.

The lesions of the skin in children differ somewhat from those which occur in adults, and these variations, both in degree and in kind, often make a differential diagnosis more difficult. Every practitioner has doubtless been struck by the similarity which at times is seen in the cutaneous lesions of the various forms of erythema to such diseases as syphilis, scarlet fever, and erysipelas. I have known the delicate pink of an abdominal erysipelas in a young infant mistaken so completely for scarlet fever that the precaution of removing the carpet in the room had already been taken. In like manner a slight grade of the efflorescence of scarlet fever may be mistaken for that of erythema neonatorum. I have also seen a harmless papular erythema closely simulating and mistaken for one of the papular efflorescences of syphilis. The efflorescences which follow the administration of antitoxin also closely simulate scarlet fever, measles, and the different forms of erythema.

Another rule, and one of equal importance, is that no single dermal lesion, whether it be a macule, a papule, a vesicle, or a pustule, makes it justifiable for us to decide that an especial disease is present. We must remember that the same cutaneous lesion may appear in almost any disease, and that it is the combination of dermal lesions and general symptoms which makes up the entire picture of the disease and justifies us in making a diagnosis.

It is very important, therefore, to have a fair, general knowledge of the local diseases of the skin as they appear in children, in order that a correct differential diagnosis can be made from the constitutional diseases with dermal lesions, which have to be treated by those who practise among children.

SCABIES.

Scabies represents the purest type of a primary disease of the skin. It is caused by an especial parasite, the *Acarus scabiei*. The following case illustrates the disease:

The child, two and a half years old, was healthy and well developed. For two weeks it had been very irritable, and its mother brought it to the hospital to inquire about an efflorescence which had appeared on its skin. On investigating the lesions a number of small papules and a few pustules scattered irregularly over the arms and chest, and one or two small pustules on the soles of the feet were found. The fingers were not especially affected, but in one or two places at the base of the fingers the efflorescence could be plainly seen. In addition to the papules and pustules there were numerous lesions of the skin caused by scratching. On the delicate skin of the abdomen was a minute black line with a vesicle at one end of it. On removing carefully with a needle a little of the fluid in this vesicle and placing it under the microscope, the parasite, which evidently had its habitat in the vesicle, could be seen. This organism, which I shall not describe more fully, as it is best studied in works especially devoted to diseases of the skin, is called the *Acarus scabiei*, and is the cause of this special dermal lesion. The black line represented the burrow by which it enters and through which it travels as far as the vesicle, where it lodges and produces irritation, causing first a minute papule, and then a minute vesicle. Finally the vesicle may become pustular. In this case, the child's mother showed the lesions of scabies between her fingers.

In contradistinction to the effects of the *Acarus scabiei* on the skin of adults we find in infants and young children that the parasite may attack the soft skin of the soles of the feet, while in the adult we do not find the lesions on the soles, as in walking the skin has become toughened in that locality. In adults efflorescences on the soles of the feet and the palms of the hands are rather unusual unless they are connected with syphilis or artificial eczema. Infants and young children are usually infected by the *Acarus scabiei* from sleeping in the bed with some adult who has scabies.

Treatment.—In the treatment of this disease it is very important to treat it in the mother as well as in the child. The clothes of the bed, of the mother, and of the infant should first be thoroughly steamed, in order to kill the parasite, and it should be impressed upon the mother that the treatment must be carried out very carefully, and that all the clothes which have come in contact with the skin must be thoroughly cleansed.

The treatment of scabies in the child should differ somewhat from that which is employed when the disease occurs in the adult, because the skin of the former is much more sensitive than that of the latter. The severe remedies which can properly be used in treating the adult should not be employed in the treatment of infants and young children.

A simple and effective ointment recommended by Bowen is as follows:

Prescription 54.

Metric.	Gramma.	*Apothecary.*	
℞ Balsami Peruviani,		℞ Balsami Peruviani,	
Petrolati....................	āā 60	Petrolati......................	āā ℥ii.
M.		M.	

For older infants and children an ointment containing some sulphur may be employed without much danger of irritating the skin.

PRESCRIPTION 55.

Metric.	Gramma.	*Apothecary.*	
℞ Sulphuris sublimati..........	7.5	℞ Sulphuris sublimati..........	ʒii;
Balsami Peruviani,		Balsami Peruviani,	
Petrolati.................... āā	30.0	Petrolati.................... āā	℥i.
M.		M.	

In the use of either of these ointments the following technique should be employed. The child is to be first thoroughly washed with warm water and soap. The skin is then dried, and the ointment is applied over the whole body, avoiding the head, which is seldom attacked by the parasite. The face especially might be irritated by the ointment. The ointment is allowed to remain on the child during the night, and in the morning is washed off with warm water and soap. The skin is then thoroughly powdered with the zinc and starch powder mentioned on page 98. This treatment is continued for three or four days, and then, if the disease is not entirely cured, it can be repeated for a few days more.

A certain amount of eczema usually follows the treatment, owing to the irritation produced by scratching, which is very difficult to prevent. This eczema should be treated by soothing applications.

PEDICULOSIS.

A parasite whose nidus is on the head appears quite frequently in children as well as in adults. It is especially met with among the poor and ill cared-for. This parasite, the *pediculus capitis*, causes extreme irritation of the skin, which often results in eczema and in enlarged glands. Although the pediculus itself is in the hair, yet by its irritating action on the scalp of the child it frequently gives rise by reflex influence to patches of eczema grouped about the nose and ears.

TREATMENT.—In treating these cases the hair and scalp should first be saturated with petroleum. This application is allowed to remain on the head for several hours, and later is thoroughly washed off with soap and water. The nits should then be carefully removed by means of a fine comb wet with vinegar. It is usually necessary to repeat the treatment for two or three days.

IMPETIGO CONTAGIOSA.

Impetigo contagiosa is a disease which usually occurs in children, but it may be found in adults. It sometimes appears as an epidemic, and in these cases, in all probability, is caused by the same micro-organism as in the isolated cases. It is usually met with among the poorly cared-for, but it may attack the healthy as well as the sick and weak.

SYMPTOMS.—The form of the efflorescence is variable. Beginning as small vesicles, the lesions soon spread over a larger area, coalesce, usually form pustules, and later become rapidly covered with a thick yellowish crust. The lesion may occur on any part of the body, but is especially common on the face and hands. The itching is very slight in these cases, and there is no constitutional disturbance caused directly by the parasite. In accordance with the idea that it is of parasitic origin, the prognosis is favorable, and the disease can usually be cured in a week or ten days.

TREATMENT.—The treatment of impetigo contagiosa is very simple, and consists in cleanliness, exposure to sunlight, and the application of an ointment such as the following:

PRESCRIPTION 56.

Metric.	Gramma.	*Apothecary.*	
℞ Acidi borici	3 75	℞ Acidi borici	ʒi;
Adipis	30 00	Adipis	℥i.
M.		M.	

FURUNCULOSIS.

Closely connected with impetigo contagiosa is furunculosis, which is supposed to be caused by the same micro-organisms that give rise to impetigo contagiosa, but which affects a different part of the skin, such as the deeper portions of the hair-follicle, in contradistinction to the upper layers of the skin, the part affected by impetigo contagiosa. These micro-organisms are called the "pus organisms," and are usually represented by the staphylococcus pyogenes aureus.

TREATMENT.—The treatment should be with an antiparasitical ointment or solution preferably containing boracic acid. In many cases in addition to this local treatment some form of constitutional treatment should be employed, as the children are usually in an abnormal condition. The lesions should be bathed every day with the following solution:

PRESCRIPTION 57.

Metric.	Gramma.	*Apothecary.*	
℞ Acidi borici	15	℞ Acidi borici.	℥ss;
Aq. destil.	240	Aq. destil.	℥viii.
M		M.	

After the parts have been thoroughly bathed with this solution the following ointment should be spread on linen compresses and applied to the lesions:

PRESCRIPTION 58.

Metric.	Gramma.	*Apothecary.*	
℞ Acidi boraci	3 75	℞ Acidi borici	ʒi;
Petrolati	30 00	Petrolati	℥i.
M.		M.	

MOLLUSCUM CONTAGIOSUM.

Another probably parasitic disease which is rare, but which is more frequent in children than in adults, is molluscum contagiosum. It occurs most commonly on the face, although it may be found on other parts of the body. The lesions consist of small, firm nodules of a whitish color, with a central depression from which matter of a sebaceous consistency may be pressed. The diagnosis is not difficult for one who has once seen the efflorescence, the only condition with which it might possibly be confused being verruca, which, however, does not occur commonly on the face, has no central depression, and does not contain any substance which can be squeezed out.

TREATMENT.—The treatment of these lesions is to puncture them, squeeze out their contents, and dress them with the same ointment as in Prescription 58.

SEBORRHŒA CAPITIS OF INFANTS.

Seborrhœa capitis consists of a collection of brownish-yellow crusts formed by a mixture of sebaceous matter and epithelial scales on the top of infants' heads. It is simply a tendency to over-production by the sebaceous glands of their secretion, which, mixed with dirt, produces this condition. The whole scalp of the infant should be perfectly clean, and in all cases it is safe and best tc remove the crusts, as they are frequently the starting-point for eczema. Plate III., B, facing page 84, represents this condition. The crusts should be gently and gradually removed by first soaking them with warm sweet oil, and then washing them off with soap and warm water. Some simple ointment should be kept continuously on the part affected, to prevent the reaccumulation of the crusts until the tendency has ceased.

TINEA TRICOPHYTINA.

The disease called *tinea tricophytina*, or *ringworm*, occurs clinically in two forms. The first form affects the scalp, and is called *tinea tonsurans*. The other form attacks the non-hairy portions of the body, and is called *tinea circinata*.

The disease itself is called *tinea tricophytina*, and the parasite which causes it is called the *tricophyton tonsurans*.

Tinea tricophytina has the peculiarity of not appearing on the scalp except in children, but is the same disease that occurs in adults in various localities, as on the face in men, destroying parts of the beard. It may also occur on any part of the body both in children and in adults. Its cause can usually be traced to the same parasitic affection in some other person or some animal.

The second form of tricophytina, *tinea circinata*, may at times appear as numerous multiple lesions in different parts of the body, and is easily affected by anti-parasitic applications.

TREATMENT.—The treatment of this disease should be active, and it is usually necessary to continue it for a long time, especially in cases in which the parasite has attacked the head. The treatment should be with the following ointment:

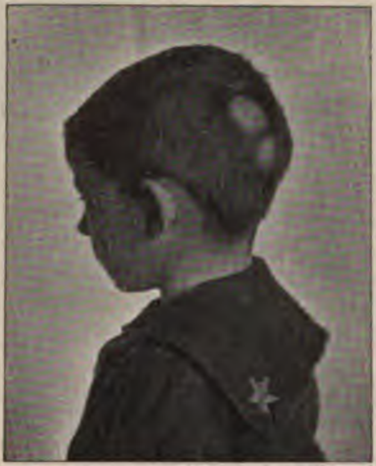
PRESCRIPTION 59.

Metric.	Gramma.	*Apothecary.*	
℞ Acidi salicylici,		℞ Acidi salicylici,	
Sulphuris	āā 3 75	Sulphuris	āā ʒi;
Lanolini	30 00	Lanolini	℥i.
M.		M.	

It should be applied twice daily, and should be thoroughly rubbed into the bald spots, the skin first having been washed with soap and water.

When the case proves to be somewhat intractable, still stronger applications can be used, and, if necessary, a certain amount of carbolic acid can be mixed with the ointment, from one-half to one drachm to the ounce of ointment.

Fig. 96.



Tinea tonsurans. Male, 8 years old.

Fig. 96 represents a boy, eight years old, with two bald spots on the back of his head. The hair over the rest of his head was thick, and there were no appearances of loss of hair anywhere else on his scalp. The areas of scalp attacked by this disease vary in size. In this special case, however, the spots were about 2.5 cm. (1 inch) in diameter. As a rule, they have a fairly regular circumference. On examining the spots one will notice that there are little short hairs on their surfaces, which evidently have broken off from lack of nutrition. On the edges of the spots this is especially noticeable. If one of the hairs is placed under the microscope, one will find a specific organism which has been determined to be the cause of this disease. It is of vegetable origin, and consists of masses of spores composed of threads of mycelium, some long and some short, which are divided into numerous segments.

TINEA FAVOSA.

Tinea favosa, or favus, is a parasitic disease. Its favorite seat is the scalp, although it may attack any part of the body. It appears in the form of small, bright yellow, cup-shaped crusts, which upon their removal leave a permanent but superficial cicatrix. These yellow crusts penetrate the hair-follicle and destroy the growth of the hair. When placed under the microscope they are found to consist almost entirely of mycelium and spores of the form called *Achorion schoenleinii*. The crusts often become confluent, forming a large thick covering over an extensive area.

TREATMENT.—The treatment is the application of an ointment to soften and remove the crusts, epilation, and anti-parasitic ointments such as described for ringworm.

TINEA VERSICOLOR.

This is a very rare disease in children. A case seven and a half years old has been reported. It is a parasitic disease caused by the microsporon furfur. The lesions consist of patches of fine yellowish scales, usually situated on the trunk. There are no subjective symptoms. Paraciticides combined as in Prescription 55, page 357, remove the disease very quickly, but it is prone to recur.

ALOPECIA AREATA.

The nature of the disease alopecia areata has not yet been determined. The lesion of alopecia areata consists of an irregular surface of the scalp entirely free from hair up to where the long hair begins to grow on its edges. The appearance of the skin over this spot is normal.

The diagnosis is made by finding a bald spot on the head having the appearance just described. The remaining part of the scalp is found to be in a healthy condition and well covered with hair.

Alopecia areata is to be differentiated especially from tinea tricophytina. In contradistinction to the sound and healthy-looking skin of the former, we find in the latter numerous short hairs, which are broken off through the action of the parasite.

Alopecia areata is somewhat intractable to treatment and runs a rather long course, but, as a rule, in children can be cured.

Treatment.—The treatment is the continual application of stimulating remedies, such as the following ointments of sulphur and tar:

Prescription 60.

Metric.	Gramma.		*Apothecary.*	
℞ Sulphuris	3	75	℞ Sulphuris	ʒi;
Petrolati	30	00	Petrolati	℥i.
M.			M.	

Prescription 61.

Metric.	Gramma.		*Apothecary.*	
℞ Olei cadini	3	75	℞ Olei cadini	ʒi;
Petrolati	30	00	Petrolati	℥i.
M.			M.	

These remedies should be used so as to produce a slight rubefaction, but not inflammation.

PEMPHIGUS NEONATORUM.

In addition to the true pemphigus of adults, the pemphigus which is secondary to diseases of a debilitating nature, and the epidemic pemphigus infantilis, we at times meet with a form of pemphigus which seems to be caused by a parasite of the skin. These cases have been described by Blomberg, but they have not yet been fully accepted by dermatologists, and it is well to remember that on the delicate skin of infants and young children impetigo contagiosa may cause the lesion of pemphigus through the activity of the parasite and the great vulnerability of the skin.

PEMPHIGUS.

Pemphigus is a disease of a constitutional character, and is represented by large blebs and bullæ. It occurs at times in infants and children as it does in adults, but is very rare. There is a form of pemphigus, however, which I have met with in infants and children in which bullæ of various sizes appear upon the limbs and trunk, and which is not connected with syphilis. It usually occurs in poorly nourished children, and can come not only as a disease of itself, but also as one of the sequelæ of debilitating diseases, such as pneumonia, rheumatism, and others. When it is secondary to other diseases it represents a condition of malnutrition, and in all probability is not connected with the real disease pemphigus. In my experience this class of cases is not especially serious, but merely represents a greater or less degree of lack of vitality of the skin.

This form of pemphigus, in which the efflorescence is secondary to other diseases, is not usually seen upon the soles of the feet or in the palms of the hands, and this is of considerable aid in distinguishing the disease from the bullous form of syphilis.

TREATMENT.—There is no especial local treatment which appears to benefit this condition of the skin, but it soon disappears when the general nutrition of the child has again become normal under appropriate feeding.

EPIDEMIC PEMPHIGUS INFANTILIS.

Where pemphigus occurs as an epidemic among infants in foundling hospitals it is of a more serious nature, and is accompanied by constitutional symptoms, represented by fever, sometimes lasting from three to six weeks. In these cases it is usually acute, but it may become chronic, and last, with intervals of recurrence, for many weeks or months. These cases are more apt to be fatal than the other forms. The true epidemic form of *purulent pemphigus*, as it has been called, is almost always fatal, and in cases in which it is not secondary to any other disease has a grave prognosis. Many of the reported cases of this epidemic form, as well as of the other forms of pemphigus, may really be only manifestations of the staphylococcus invasion.

DERMATITIS EXFOLIATIVA NEONATORUM (Ritter's Disease).

In the year 1878 Ritter gave the first complete description of the disease *dermatitis exfoliativa neonatorum*. Previous to this date cases of this affection had been reported, but many of them were regarded as some rare or unusual manifestation of pemphigus. Ritter studied and reported the cases which he saw at the Foundling Asylum in Prague from 1868 to 1878. A careful review of Ritter's original observations of these cases has been made by Elliot. The majority of cases were in male infants, and the mortality was found to be 48.82 per cent. The disease is very rare. It occurred rarely before the end of the first week, and usually appeared between the second and the fifth week of life. It was found to vary greatly in the intensity of its symptoms. In some cases a dry scaly condition of the skin preceded the subsequent lesions, which had apparently lasted after the physiological desquamation of the epidermis had taken place.

Symptoms.—The first symptom noticeable in these cases was a diffuse redness, usually over the lower half of the face about the mouth, sometimes, however, beginning in some other portion of the body, and at times being universal from the beginning. This hyperæmia of the skin spread rapidly, and in a few days became universal, the extremities, as a rule, being the last parts affected. The mucous membrane of the mouth and nose was at times affected, and the conjunctivæ usually participated in the hyperæmia. The color of the efflorescence varied from a light to a dark purple-red. As the hyperæmia extended to new surfaces, those which were first affected began to desquamate. This desquamation at times gave no evidence of exudation, the epidermis being simply thickened, and the loosened epithelium separating easily. At times other lesions appeared, such as milia, and sometimes the horny layer of the skin was raised above an intensely reddened base, and large, irregularly shaped bullæ filled with fluid were formed. After the desquamation had taken place the skin recovered its normal condition, occasionally very rapidly, but it remained for some time rough and irritable. In the cases in which there was no exudation a longer time was necessary for the separation and regeneration of the epithelium.

Usually the disease was found to run its course in from seven to ten days. Relapses were sometimes observed ten or twelve days after the first attack, but were always mild.

In typical cases the process was unaccompanied by any fever or systemic disturbances unless some complication existed. The functions were normal, and the weight of the infant remained stationary or was even at times increased. The fatal cases resulted either from the intensity of the attack or from some intercurrent affection or sequela, such as furunculosis. The disease is usually recognized as a local septic infection of the skin, and it would seem that it should be distinguished from the pemphigus which occurs in the early weeks of life.

I have myself seen but one case in which it seemed that this diagnosis of dermatitis exfoliativa could reasonably be made.

This case was a male infant, who at the fourth or fifth day of its life presented a marked condition of erythema neonatorum. After a few days this erythema began to desquamate slightly, but somewhat later a pronounced dermatitis appeared and ran its course for a week. During the course of the disease there were lesions of various kinds represented by a few pustules and bullæ, but mostly by an intense erythema. The lesions gradually grew less intense, a profuse desquamation took place, and the skin then presented a normal appearance. During the course of the disease the infant did not show any constitutional symptoms, and gained somewhat in weight. The parents were healthy, strong people, with good hygienic surroundings.

DERMATITIS.

Dermatitis is an inflammatory affection of the skin, produced by some recognized cause. The lesions are, as a rule, fugitive, and with a few exceptions are not characterized by an especial and peculiar form of efflorescence. The course of the disease, and the recognition of some definite exciting cause, enables us to distinguish this condition from others which resemble it.

Clinically the group may be divided into a *dermatitis traumatica*, *dermatitis venenata*, *dermatitis calorica*, and *dermatitis medicamentosa*.

Dermatitis Traumatica.—Dermatitis traumatica is the term applied to the local reaction which takes place in the skin, following some trauma, either slight or severe, such as may result from pressure, friction, or direct blows. The lesions vary from a simple, temporary erythema to deep and extensive ulcers, as in certain bed-sores.

Dermatitis Venenata.—Dermatitis venenata is the name applied to those dermal lesions which are caused by the external application or contact of irritating substances. The inflammatory condition may be due to simple *mechanical* irritation, such as is sometimes caused by the lodgement in the skin of small particles of matter; or it may be due to a *poison*, arising either from emanation of a poisonous volatile principle or from actual contact. *Ivy poisoning* is one of the most common and important examples of this class of affections. It is caused by contact with the leaves of the *rhus toxicodendron*. The cases are most common in the autumn, probably owing to the fact that people are tempted to gather the leaves because of their brilliant coloring at this season of the year. There is some difference of opinion as to the volatility of the poisonous active principle. *Rhus venenata*, or poisonous oak, is another common cause of ivy poisoning.

Some of the more important irritants which may produce a dermatitis venenata are *chrysarobin*, an effective remedy in psoriasis, preparations of *carbolic acid*, *turpentine*, *iodine*, *mercury*, and *sinapis*. There are sixty or more drugs (White) cited as more or less frequent causes of a dermatitis. Knowing the great delicacy of an infant's skin, we cannot be too cautious in prescribing ointments and applications, or in seeking for the cause of a dermatitis in some article which is of common use perhaps, but at times an undoubted source of irritation to the skin.

SYMPTOMS OF IVY POISONING.—The eruption appears generally within a day or two after exposure, with redness, œdema, and papules, which pass rapidly to the stage of vesicles, which may become pustular from a secondary infection. The course of the disease is from two to six weeks. The face, hands, and genitals are especially liable to be the seat of the disease, the extension to other parts of the body taking place by means of the hands.

DIAGNOSIS.—The lesions of ivy poisoning resemble those of an acute eczema. In making the diagnosis especial attention should be paid to a history of exposure to ivy, the time of year, the asymmetry in the distribution of the lesions, and the history of previous attacks, and especial susceptibility will often aid in the exclusion of other similar conditions, especially eczema.

TREATMENT.—There is no specific remedy. The treatment is the same as in an acute eczema, except that it is well to begin by washing the skin thoroughly with soap and water. Prescription 66, page 370, may be used as a wash, and some simple ointment, as Prescription 68, page 372, may be applied.

Dermatitis Calorica.—Dermatitis calorica is a form of dermal inflammation produced by the milder degrees of heat. The action of the sun's rays is the simplest and most common type met with in children. The lesions vary from a simple erythema to vesicles and bullæ, depending upon the intensity of the heat, the duration of exposure, and the susceptibility of the skin in an individual case. Diminution of heat may produce lesions the character and course of which are very similar to those which result from an excess of heat. *Chilblains* are a common example of the result of a diminution of heat. They occur chiefly in children with feeble circulation who wet or chill their feet, and suddenly heat them. In such instances the skin is red, soft, and boggy. The boggy areas may break down and form indolent ulcers, which are difficult to cure.

TREATMENT.—The treatment of these cases of dermatitis calorica is the application of some simple lotion, as in Prescription 63, page 367, or of an ointment, as in Prescription 68, page 372.

Dermatitis Medicamentosa.—Dermatitis medicamentosa is a general term including a great variety of lesions produced by the action of certain drugs administered internally. *Arsenic* may produce an efflorescence of a very complex character which may be erythematous, papular, urticarial, bullous, pustular, and even hemorrhagic. It may also give rise to a herpes zoster, as a result of certain changes in the nerve-endings. *Belladonna* often produces an efflorescence of an erythematous character resembling closely that which occurs in scarlet fever. The *iodides* are especially likely to cause an efflorescence of *acne*. The *bromides* often cause lesions of a papular or pustular character. Both the bromides and iodides occasionally cause lesions of a most unusual character. *Chloral*, *digitalis*, *opium*, *quinine*, the *salicylates*, and many of the recent new remedies sometimes cause an efflorescence. The erythematous, urticarial, and papular efflorescences which are seen at times after the administration of *antitoxin* will be more fully described under diphtheria.

TREATMENT.—The treatment consists in discovering and discontinuing the drug the use of which is causing the irritation of the skin.

SUDAMINA.

Sudamina is a non-inflammatory condition of the skin caused by occlusion of the ducts of the sweat-glands. The lesion is represented by minute pearl-like vesicles occurring on the skin in crops, and is not apt to appear in febrile conditions. The disease is of no significance and requires no treatment.

ERYTHEMA.

Erythema plays an important part in the diseases of infants and young children. Although it is one of the most common and readily diagnosticated diseases of the skin which occur in early life, yet at times it is quite difficult to differentiate it from other diseases, owing to the variety of its forms. It may be divided into two broad classes: (1) the congestive form, or *erythema simplex*, which is caused by traumatism and by various drugs, and is also symptomatic of the acute exanthemata; (2) the inflammatory form, *erythema multiforme*, which may affect any part of the body and either small or large surfaces. It has, however, a predilection for the backs of the hands and of the feet. Its lesions may be represented by maculæ, or in the process of its evolution these maculæ may develop into maculo-papules, vesico-papules, papules, vesicles, and even bullæ. The lesions vary in size. The color varies from bright red to purplish red, and is sometimes very vivid. The delicate texture of the skin of young subjects is more likely to show variations in the color and the form of its lesions than is the fully developed and stronger skin of the adult.

Erythema Simplex.—SYMPTOMS.—The symptoms of the congestive form are varied, and they do not accompany each manifestation of the disease with any especial regularity. The slightest local irritation, whether from parasites or trauma of any kind, changes in temperature, reflex irritation from the close connection between the digestive organs and the skin, and many other reflex manifestations, may produce the disease.

Erythema Multiforme.—SYMPTOMS.—In erythema multiforme there may be pains in the joints simulating rheumatism, malaise, slight fever, nausea, coated tongue, loss of appetite, and a swollen, tender skin. These more marked symptoms are, however, often absent, and the lesions of an erythema multiforme commonly appear on the skin of young subjects without any special general symptoms accompanying them. It is better in nursery practice not to endeavor to classify this protean disease under special names which have been handed down from time immemorial in the text-books, and which have no particular significance. They have been used indefinitely by physicians, and the same form of lesion is sometimes called by one name and sometimes by another.

TREATMENT.—The treatment of all forms of erythema is practically the same. It consists chiefly in the application of a simple powder of oxide of zinc and starch, and of a lotion consisting of either lime-water or rose-water in which calamine and oxide of zinc are suspended.

PRESCRIPTION 62.

Metric.	Gramma.	*Apothecary.*	
℞ Zinci oxidi	7|5	℞ Zinci oxidi	ʒii;
Amyli tritici	60|0	Amyli tritici	℥ii.
M.		M.	

S.—For external application.

PRESCRIPTION 63.

Metric.	Gramma.	*Apothecary.*	
℞ Zinci oxidi,		℞ Zinci oxidi,	
Calaminæ præparatæ.........āā	7.5	Calaminæ præparatæ.........āā	ʒ ii ;
Aquæ calcis	240.0	Aquæ calcis	℥ viii.
M.		M.	

S.—For external application.

ERYTHEMA INTERTRIGO.

This is a congestive form of erythema. It is represented in Plate III., A, opposite page 84. This form of erythema is that which occurs in infants in the folds of the groin, neck, and axillæ. The lesion has already been described under erythema simplex. Napkins soaked with urine and allowed to remain for some time without being changed are a frequent cause of this condition.

The treatment is to keep the skin clean and dry by the application of a simple powder such as is given above on page 366. No water should be used on the parts affected, but in its place equal parts of lime-water and water can be used for washing.

In the more severe forms of this disease, in which the erythematous condition has become eczematous, and where the skin in the folds of the groins, of the neck, or of the axillæ shows fissures and the moist condition represented by eczema madidans, I have found an application of boracic acid powder efficacious.

ERYTHEMA NODOSUM.

Another form of erythema, called erythema nodosum, is a disease which is closely allied to erythema multiforme. The general characteristics and symptoms of erythema nodosum are well represented in the following case:

A little girl, five years old, was perfectly well until two days before she came under observation. At that time she began to have loss of appetite, fever, and malaise, followed by pain in both her legs. Following these general symptoms an efflorescence appeared in various places on her legs. It was found above and below the knees, but mostly over the tibiæ and extending down as far as the ankles. These lesions were from 1.2 to 2.5 cm. (½ to 1 inch) in diameter, and were of a somewhat irregular elliptical outline. They were of an erythematous type and had a delicate pink color. The skin over the lesions was hot in comparison with the unaffected portions of the skin around them. The lesions were tender on pressure, and their tissues were somewhat indurated, so that the feeling was that of a hard, raised swelling.

The disease is self-limited, but is irregular in its course. It usually disappears in about two weeks. Its cause is not known. The treatment is simply palliative.

ERYTHEMA URTICATUM—URTICARIA.

Nettle-Rash, Hives.—The term urticaria has been applied to an efflorescence characterized, as a rule, by wheals, which appear suddenly and disappear quickly. It is accompanied by intense itching and burning, and may show itself on any part of the skin, in lesions either small or large in number.

It is commonly caused by irritation of the gastro-enteric tract. The disease may end in two or three days, but usually lasts for some weeks, and may become chronic; it is essentially, however, an acute affection.

If the lesion has been severe there may be slight desquamation, but this is rare. Sometimes there may be only one attack; again there may be relapses, and in some forms and in certain skins it may occur from year to year.

When seeking for the cause of an outbreak of urticaria it is necessary to investigate carefully as to whether there has been an error in diet. In children some simple article of food may cause an urticaria to appear, just as in some adults the disease occurs from an idiosyncrasy which prohibits them from eating oysters, lobsters, strawberries, or certain other articles of diet. Again, in some individuals, certain drugs, such as chloral, bromide of potash, chlorate of potash, and belladonna, may cause the dermal lesions of urticaria. The wheals of urticaria frequently occur as a symptom in the course of various diseases, such as scabies, or may be caused by the bites of insects.

Treatment.—The treatment should be directed first to the removal of the cause of the dermal irritation. When this cause has been removed the dermal lesions will, as a rule, disappear, unless still further irritation has been produced by scratching the lesion or by its being too severely treated by the physician.

The diet should be milk for a time, and experiments should be made with different articles of food to see which one may cause this especial form of irritation. The bowels should be carefully regulated. The local applications consist of remedies to relieve the itching and burning, in the wearing of unirritating clothing and soft linen next the skin, and in a powder of starch and zinc, made as described on page 366, frequently applied to the lesions at intervals during the day. When the itching is extreme, anti-pruritic lotions and ointments should be used, as in the following prescriptions:

PRESCRIPTION 64.

Metric.	Gramma.	*Apothecary.*	
℞ Pulv. calaminæ	7.5	℞ Pulv. calaminæ	ʒii;
Aq. calcis	240.0	Aq. calcis	℥viii;
Acidi carbolici	1.87	Acidi carbolici	ʒss.
M.		M.	

When this lotion is not sufficient to allay the irritation and when the burning is extreme, the following ointment can be applied:

PRESCRIPTION 65.

Metric.	Gramma.	*Apothecary.*	
℞ Menthol	0.6	℞ Menthol	gr. x:
Adipis	30.0	Adipis	℥i.
M.		M.	

ECZEMA.

Eczema is a disease of the skin which plays a much greater rôle in infancy and early childhood than in any other period of life. It is often very difficult to cure. Even in the milder forms of the disease we should be cautious about giving too favorable a prognosis at first, for the disease may extend and involve new areas of skin.

PATHOLOGY.—The pathological changes which occur have been designated by certain descriptive names, such as *eczema erythematosum*, generally secondary to other lesions; *eczema papulosum*, which may be a terminal or merely a secondary stage of the process; *eczema vesiculosum*, which is never primary, but is preceded by a papular stage, and may end as such or pass on to the more advanced lesions; *eczema madidans*, or "weeping eczema," in which the vesicles form large blisters containing sero-purulent or sero-hemorrhagic contents which exude and form crusts; *eczema pustulosum*, which may begin as a primary lesion or develop secondarily to the vesicular or madidans stage; and, finally, *eczema squamosum*, which is never primary, but represents the final stage of the pathological process in which the epidermal scales are thrown off, leaving exposed a dry, itchy, reddened skin. After resolution, cicatrices rarely occur, unless from severe secondary changes.

SYMPTOMS.—Clinically the disease may be divided into the *acute* form, characterized by a sudden onset, short course, and frequent recurrence of any of the pathological conditions just mentioned; and by the *chronic* form, which represents the more characteristic lesions of cell infiltration and thickening of the skin, scaling, fissures, and ulcers. The disease may begin as an acute or as a chronic process, starting as an erythema or as isolated and grouped papules, vesicles or pustules, either singly, simultaneously, or in succession, resulting in redness, oozing, scaling, crusting, and infiltration. The intense itching and burning, without tenderness, are the important and characteristic points serving to distinguish the condition from a dermatitis. The disease may be divided into two types, *regional* or *local eczema* and *universal eczema*.

Regional Eczema.—The most common form of regional eczema occurring in infants and young children is localized on the *face*, and commonly extends to the *neck*. This form is especially distinctive of infancy, and is frequently very intractable. No one form of treatment or kind of application benefits every case, but one remedy after another may have to be tried. As the infant grows older this form of eczema passes away of itself and is not apt to return.

Many instances of local eczema produced by some irritation at or near the place affected, or perhaps in an entirely different part of the body, are met with in children. This is usually called *reflex eczema*, an example of which may be found in the irritation of the scalp, such as occurs from pediculi, from which a local reflex eczema may develop on the back of the neck.

Other varieties of regional eczema are limited to the *eyes*, giving rise t swelling, pustules, furuncles, and conjunctivitis, a condition which ma easily be mistaken for erysipelas, but shows no tenderness and itche badly. The *ears*, *genitals*, *legs*, *feet*, and *hands* may all be the site o eczematous lesions.

Universal Eczema.—Attacks of universal eczema may occur in children as they do in adults, and are often very intractable. The lesions are essentially the same as in regional eczema, but differ in that they are more universally distributed.

TREATMENT.—The importance of the treatment of eczema does not depend so much on any particular ointment or drug as on the method of applying the remedy. The principal indication is to keep the child quiet and the skin free from the irritation of scratching, thus allowing it to recover its vitality. At times it is necessary to strap the child on its back in bed and to have a nurse in constant attendance until the more irritable stage of the disease has passed off. Scratching the lesions even for a few moments may retard the recovery for many weeks.

If necessary in the early hours drugs of a soothing nature may be given to prevent undue nervous symptoms. The nurse should be gentle, and should endeavor continually to divert the child's mind. The treatment consists, then, first in allaying the itching by local applications, and of so covering the part affected that scratching is impossible. It is often necessary to pin the sleeves of the dress to the napkin in order to control the hands. The discomfort from the restraint will soon pass away, and the method of treatment is not cruel. Certain general precautions should be observed. Heat and excess of cold should be avoided, and the clothing should be thin and as non-irritating as possible. The physical condition of the child should be carefully investigated in regard to the urine, blood, bowels, appetite, and nutrition, and appropriate symptomatic treatment should be given.

Drugs.—Drugs may be administered as washes, powders, and ointments, but never as tinctures, in the acute forms of eczema. Water should never be used in the treatment of acute eczema. The list of drugs is long, and only a few of the more important ones need be mentioned. A simple powder of starch and zinc oxide, such as is given on page 366, will be found most useful on moist surfaces. In all acute cases, and as an antipruritic, the following prescription will be found of value.

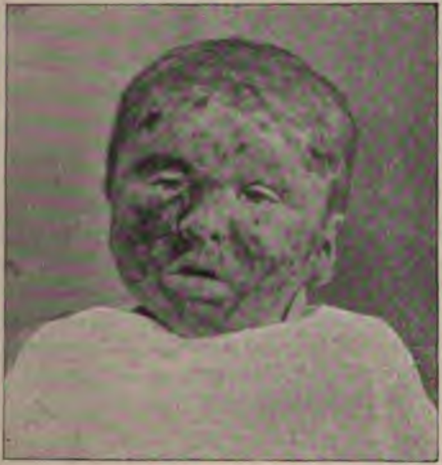
PRESCRIPTION 66.

Metric.	Gramma.	*Apothecary.*	
℞ Zinci oxidi	15.00	℞ Zinci oxidi	℥ss;
Glycerini	3.75	Glycerini	ʒi;
Acid. carbol	1.88	Acidi carbolici (xtals)	ʒss;
Aq. calcis q.s. ad	240.00	Aquæ calcis q.s. ad	℥viii.
M.		M.	

Calamine may be used in the above prescription in place of the oxide of zinc, as in Prescription 64 on page 368, and it is often desirable to add

60 c.c. (2 ounces) of camphor-water to replace an equal quantity of lime-water. An ointment of sulphur and oxide of zinc, 1.88 gramme (½ drachm) of each to 30 grammes (1 ounce) of vaseline, is very useful in eczema of the scalp.

FIG. 97.



Eczema capitis.

In the treatment of the dry, scaling, infiltrated form of chronic eczema, when there are no excoriations or acute lesions, the following prescription of White's may be used.

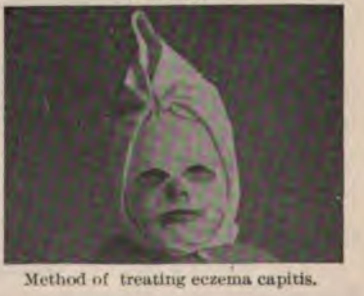
PRESCRIPTION 67.

Metric.	Gramma.	*Apothecary.*	
℞ Sapo viridis,		℞ Sapo viridis,	
Alcohol ....āā	60	Alcohol ....āā	℥ij;
Filter and add		Filter and add	
Olei cadini	15–30	Olei cadini	℥ss–j.
M.		M.	

Fig. 97 represents a case of eczema of the scalp and face which illustrates the disease very well.

The lesions were confined to the head and face. They consisted of papules, pustules, crusts, some excoriated patches caused by scratching, and a thick, rather œdematous condition of the skin, especially around the lips, nose, and eyes. The hair was cut off, and various lesions were found on the scalp; in certain parts of the scalp a reddened moist condition, called eczema rubrum, was found.

FIG. 98.



Method of treating eczema capitis.

The treatment was as follows: the crusts and the thickened tissue of the face and scalp were first softened by means of a poultice. After the larger crusts had been removed, a mask, as shown in Fig. 98, was applied to the face and scalp. The inner surface of this mask was thickly spread with the following ointment:

Prescription 68.

Metric.	Gramma.	*Apothecary.*	
℞ Unguenti zinci oxidi, Lanolini ....āā	30 00	℞ Unguenti zinci oxidi, Lanolini ....āā	℥i.
M.		M.	

An interesting complication of universal eczema which may at times arise is illustrated in the following case:

A girl came to the hospital to be treated for torticollis. The head was drawn to the left side and she could not straighten it. This condition had lasted for many months.

On examining the child I found that she had the usual universal eczema of a chronic type affecting the head, face, and extremities. On examining the neck I found a number of enlarged tender glands. These enlarged glands were evidently caused by reflex irritation from the eczema, and were the cause of the torticollis.

She was treated with the zinc oxide ointment (Prescription 68) and the usual bandage and mask, and in a short time, although the eczema was not entirely cured, the irritation in connection with it had been so much lessened that the glands of the neck gradually subsided and disappeared, and the child was able to hold her head straight.

We are often asked whether the eczema of infants is contagious. I have seen instances where the nurse who was taking care of a case of eczema developed the disease on her hands. The lesions were, however, apparently caused by washing the infant's napkins, as her hands were cured by local treatment, and the subsequent use of rubber gloves while washing the napkins prevented her from again contracting the disease. Cases of this kind give rise to the idea that eczema is contagious, but the probability is that they are simply cases of artificial dermatitis caused by irritating substances of various kinds, and that there is no especial germ which causes eczema. We can, therefore, say that the disease is not contagious, and that simple cleanliness and protection of the hands by means of rubber gloves are all that is necessary to prevent the disease being contracted.

PSORIASIS.

Nothing is known of the real cause of psoriasis. So far as we can ascertain, it is not dependent on any micro-organism. When the disease is well developed the diagnosis is very simple, and its lesions correspond, as a rule, to those which are commonly met with in the adult. It begins with small papules, which almost immediately become covered with scales. These scales have a pearly white color, and on removing them we find a bleeding surface, showing that they are more closely connected with the corium than is the case in other diseases in which desquamation takes place, such as dermatitis or scarlet fever.

The efflorescence of psoriasis is general, and is, as a rule, marked on the elbows and knees, for in these places the lesions coalesce and the scales are especially thick.

I have noticed in the psoriasis of children that the type of the disease is often so mild that we can scarcely believe we are dealing with the same affection that we are accustomed to see in the adult. In some cases a few lesions scattered here and there, especially on the back over the scapulæ, will be all that represent the disease, and are easily cured, even disappearing of themselves in a few months. Besides affecting the trunk and extremities, the efflorescence may occur on the scalp, especially along the edge of the hair on the forehead, but the disease is not very common on the face. Psoriasis is apt to recur even at intervals of years, so that we cannot say that it can be absolutely cured, although at times it may disappear under treatment and never return.

TREATMENT.—The treatment of psoriasis in children should be milder in form than that employed in treating the adult. In the above case an ointment of chrysarobin applied to the lesions in the evening and washed off with soap and water in the morning was used, there being no treatment during the day.

PRESCRIPTION 69.

Metric.	Gramma.	*Apothecary*	
℞ Chrysarobini	0|60	℞ Chrysarobini	gr. x;
Petrolati	30|00	Petrolati	℥i.
M.		M.	

This ointment stains the skin, but not permanently. It should never be applied to the face or the scalp, and should be used with great care, as it causes on some skins considerable irritation, and at times a severe dermatitis. With ordinary caution, however, this need not occur.

In intractable cases in which this milder form of ointment is not efficacious, the strength may be increased to 1 or 1.5 grammes (15 or 20 grains) to the ounce.

It should be remembered that chrysarobin stains the clothes black indelibly, so that old sheets and night apparel should be used while the treatment is being carried out.

In place of this ointment you can use on especially irritable skins, or on the face and scalp, the following prescription of sulphur and tar:

PRESCRIPTION 70.

Metric.	Gramma.	*Apothecary.*	
℞ Sulphuris	3|75	℞ Sulphuris	ʒi;
Olei cadini	1|87	Olei cadini	ʒss;
Adipis	30|00	Adipis	℥i.
M.		M.	

PRURIGO.

Prurigo occurs in two forms in infants and children,—(1) *prurigo mitis infantilis* and (2) *prurigo ferox*.

(1) **Prurigo Mitis Infantilis.**—Prurigo mitis infantilis occurs in infants two or three months old, and may last for some years. It is closely allied

to papular erythema, but is more chronic and has a greater tendency to recur. It is very rare in America.

Symptoms.—It begins with little nodular infiltrations, especially marked on the anterior surface of the extremities, and is accompanied by great itching. It may appear on the face. It does not lead to an infiltration of the skin or to the formation of pus.

Treatment.—The treatment consists in remedies to relieve the itching and allay the eczema with which it is usually complicated.

(2) **Prurigo Ferox.**—Instead of this mild form a more severe type of prurigo occurs at times. This latter form is far more serious in its symptoms and in its prognosis, and may continue through life. The disease, which is characterized by the same dermal lesion as that just described, is progressive from the beginning; it usually starts on the legs, and the skin becomes thicker as it descends. The efflorescence is accompanied by enlarged glands, especially in the inguinal region.

The disease is rare in America, but is common in Germany.

Its etiology is very obscure, and it is a most intractable chronic affection.

Treatment.—The treatment is palliative.

For the extreme itching caused by the papules an application of the following ointment may be used:

Prescription 71.

Metric.	Gramma.		Apothecary.	
℞ Unguenti diachyli,			℞ Unguenti diachyli,	
Petrolei	āā 30	00	Petrolei	āā ℥i.
M.			M.	

S.—To be applied on flannel three times a day for ten minutes, and to be followed by the application of this ointment:

Prescription 72.

Metric.	Gramma.		Apothecary.	
℞ Unguenti diachyli,			℞ Unguenti diachyli,	
Petrolati	āā 30	00	Petrolati	āā ℥i.
M.			M.	

If there is much infiltration, sapo viridis should be applied at night and washed off the next morning. It must, however, be used with caution, as it is very irritating.

HERPES ZOSTER.

Herpes zoster is a disease which affects both children and adults. The cause of the disease has not as yet been determined, but it is a condition closely connected with the nerves.

Symptoms.—The general symptoms of herpes zoster are fever, loss of appetite, and pain in some part of the head, trunk, or extremities. The pain is always located in the course of certain nerves. In some cases, however, the pain and constitutional symptoms are absent.

One of the characteristics of the efflorescence is that, as a rule, it is unilateral. It is extremely rare for the affection to be bilateral and to extend around the body. Cases of this kind, however, have occurred, and do not seem to be any more severe, except that larger surfaces are affected, than where the affection is unilateral. The character of the efflorescence is essentially vesicular, and is to be differentiated from varicella, which might be accompanied by the same general symptoms and is also essentially a vesicular disease. The efflorescence of varicella is general, is not limited to any special distribution of the nerves, nor is it painful, while the efflorescence of herpes zoster is limited to the distribution of a special set of nerves. The vesicles become somewhat pustular, and soon crusts are formed.

The disease runs a definite course of about fourteen days, and from the beginning is accompanied by considerable pain, although according to my observations the pain is not so severe in children as in adults. nor is the itching so annoying.

DIAGNOSIS.—The diagnosis of this disease is very easily made from the general symptoms of pain, fever, and malaise, in combination with the characteristic efflorescence, and we at once know with what disease we are dealing, for no other affection of the skin has so definite a distribution.

PROGNOSIS.—The disease is usually benign in children, but the case of a child four years old who died during an attack of herpes zoster without other assignable cause has been reported.

TREATMENT.—The treatment is simply palliative. What I am accustomed to do is to regulate carefully the child's diet, as I would in any disease with general constitutional symptoms, and to endeavor by the application of lotions to allay the pain. Sometimes merely a simple powder, such as the prescription on page 366, is sufficient to allay the local symptoms.

PITYRIASIS.

Pityriasis is a term that is now, like the word lichen, seldom used without an accompanying adjective. There are two recognized forms of the affection.

(1) **Pityriasis Rubra** is a rare disease in children, characterized by hyperæmia and fine scales affecting, as a rule, the whole cutaneous surface. It may be attended with great constitutional disturbance and lead to death. Its duration is always uncertain.

(2) **Pityriasis Maculata et Circinata,** or **Pityriasis, Rosea** affects children as well as adults. It appears in the form of small patches of scales scattered over the trunk, legs, and arms. These patches either spread peripherally or unite to form larger patches while the centre undergoes involution; we thus see a reddish scaling border and a characteristic yellowish centre. There may or may not be great pruritis accompanying it. In Vienna this affection is still regarded as a form of ringworm, a position that cannot, however, be maintained. Its etiology is obscure. It gets

well spontaneously in from two to ten weeks, and is best treated by mil soothing, and anti-parasitic applications.

VERRUCÆ.

Verrucæ (warts) are circumscribed outgrowths of the papillæ of th skin with an accompanying increase in the thickness of the epidermi layers. They are common in children, especially on the hands, and th old view that they are contagious and auto-inoculable has gained man adherents of late. They are of various aspects and shapes, and may b treated locally, as a rule, with success, although some are quite obstinate The most efficacious method of treatment is to paint each wart with solution of salicylic acid in flexible collodion (Prescription 73).

PRESCRIPTION 73.

Metric.	Gramma.	*Apothecary.*	
℞ Acidi salicylici	3|75	℞ Acidi salicylici	ʒi.
Collodii	30|00	Collodii	℥i.
M.		M.	

This is applied with a camel's-hair brush twice a day for three days. Then it is soaked off by prolonged bathing in warm water, with the addition of pumice soap if there is no inflammation. This will usually remove a portion of the wart, and the process should be repeated as long as any of the growth is left.

The treatment with salicylic acid is not always successful, and recourse may then be had to glacial acetic acid, or to some other caustic, carefully applied; or the growth may be excised.

LENTIGO.

Lentigo (freckles) is a small aggregation of pigment deposited in the skin, and is commonly seen in children of ten years and upward, especially in those of light complexion. They are usually situated on the face and hands, but may occur on the covered portions of the body, a fact that led Hebra to regard them as not due to the action of the sun. There can be no doubt, however, that the sun is the chief agent in their production. Their removal is often difficult and requires the use of strong irritants, such as corrosive sublimate. It is rarely advisable to attempt their removal in young children.

MELANODERMA LENTICULARIS PROGRESSIVA.

Melanoderma lenticularis progressiva (Kaposi's disease) is a very rare disorder, and is seldom met with in this country. In this affection spots of pigment like freckles appear first on the uncovered parts of the body, finally extending more or less over the whole cutaneous surface. The pigment-spots are the first lesions seen, but later an atrophy of the skin and the formation of small angiomata dotted over the surface take place, giving the child an extraordinary appearance. The disease is usually found in more than one child in the same family, and its etiology is very obscure. Malignant tumors with a fatal ending often result from this affection. I have had two cases under my observation for four or five years. One has lately died, and the autopsy showed no metastases in the interal organs, the disease proving purely a local affection of the skin; the other has returned to the hospital in a worse condition than ever before. No treatment of any kind, medical or surgical, has had any effect on this case.

LICHEN.

Many of the affections that were formerly included under the head of lichen are now considered by most authorities to belong in other groups, notably in that of eczema. A diagnosis of lichen is never made by American dermatologists, but *lichen planus* is a well-marked skin disorder which retains a place of its own. It rarely occurs in children, but when present it follows about the same course as in adults. It is characterized by firm papules of an irregular shape and glistening appearance, of a peculiar reddish-blue or violet color, with usually a slight depression in the centre. The individual papules may coalesce, so as to form patches of greater or less extent, covered with fine scales. It is often accompanied by great itching and discomfort. It attacks all parts of the body, showing a predilection, however, for the flexor surfaces of the arms and legs. It may last for many months, and in the most favorable cases does not disappear for several weeks. The general health is not usually affected, except by the exhaustion that may be caused by intense itching. It may be confounded with a papular syphilide, which it often closely simulates, and sometimes it may be mistaken for an eczema. Arsenic is of value in chronic cases, and anti-parasitic lotions and ointments, especially those containing tar in some form, give relief as external applications.

ICHTHYOSIS.

The disease *ichthyosis* as it occurs in infants and young children does not differ in its general pathology from that which is seen in adults. It may occur in intra-uterine life, and is then designated *fœtal ichthyosis*.

The most thorough work which has been done on the ichthyosis of infancy and childhood is that of Ballantyne of Edinburgh, who designates that form which has occurred in utero and is fully developed at birth as (1) *fœtal ichthyosis*, while the form which begins in the early weeks of infancy he speaks of as (2) *ichthyosis neonatorum*.

(1) **Fœtal Ichthyosis.**—The severity of fœtal ichthyosis varies greatly.

(*a*) *Severe Form.*—The grave form, according to Ballantyne, is developed probably about the fourth month of intra-uterine life, and is characterized at the time of birth by the existence all over the body of horny epidermic plates separated from one another by fissures and furrows, associated with deformities of the mouth, nose, eyes, lips, and limbs, and leading within a few days or even hours to the death of the infant. As in most cases infants with this disease are born alive, fœtal ichthyosis cannot be considered to be a cause of intra-uterine death. The disease does not seem to affect especially the size and weight of the infant. As a rule, the viscera at the post-mortem show nothing abnormal except an unusual degree of congestion. The microscopic examination shows no extension of the keratinizing process on any of the mucous membranes, and the disease is an abnormality in the development of the skin, there being an excessive proliferation of the layers of the epidermis.

Symptoms.—In the early hours of life infants with this disease usually cry loudly and continuously, but sometimes the cry is feeble and often very peculiar. The respiration is usually impeded by the blocking of the nostrils with epidermal masses. Suction is rendered difficult or altogether impossible by the presence of ichthyotic plates around the mouth. They, are, however, usually able to swallow readily. As a rule, nothing abnormal is found in connection with the urine or the fæces. Insomnia is a marked symptom.

These infants have a very repulsive appearance, and there is a cadaveric odor arising from the abnormal condition of the skin. This ichthyotic condition of the skin is usually universal, but is most evident upon the face. The mouth is ordinarily kept open by the contraction of the surrounding parts, and from its angles radiate fissures which simulate the rhagades of syphilis. The lips are thick and everted, so as to form an irregular entrance to the gaping buccal cavity. The chin is receding. The nose can scarcely be seen, it is covered so thickly with the epidermal plates around the nostrils. There is usually ectropion of both eyelids, but sometimes only of the upper one, the orbits seeming to be occupied by fleshy tumors. If, however, we separate the swollen eyelids, the normal eyeball is found to lie beneath. The external ear seems to have disappeared almost entirely.

In contradistinction to the opinion formerly held that fœtal ichthyosis was a general seborrhœa, it is now generally supposed to be connected with the disease as it occurs in the adult.

Prognosis.—The prognosis of the disease is almost always unfavorable.

Treatment.—The treatment should be active and directed towards softening the epidermic scales by means of warm oil inunctions.

(*b*) *Mild Form.*—Besides the grave form of fœtal ichthyosis, there is a much milder form of the disease. It develops during intra-uterine life, and shows a continuous layer of a substance resembling collodion extending over the whole body and falling off in small flakes resembling pieces of tissue-paper. These general appearances are sometimes accompanied by ectropion and eclabium. The disease is not, as a rule, fatal, and often terminates in complete or partial recovery. There have not been any instances, so far as I know, of an infant being born dead with this form of ichthyosis.

Treatment.—The treatment of this second form should be by continual stimulation of the child's general strength and by great care of the skin.

(2) **Ichthyosis Neonatorum.**—Ichthyosis in the new-born infant, in whom at birth there was no sign of the disease, may occur. It presents the same appearances as the milder form of fœtal ichthyosis and the ichthyosis of the older child and the adult.

This is the common form of ichthyosis, which occurs at all ages. It begins for the most part in the early months of life, is essentially chronic, and is very intractable to treatment.

Treatment.—It should be treated by the administration of a warm bath once daily, followed by an inunction with glycerite of starch.

SCLERODERMA.

Scleroderma is a disease which at times occurs in children as it does in adults. It consists of an induration of the skin either in bands or in patches, or is diffuse, having a board-like hardness, so that the skin cannot be raised by the fingers and feels as though it were tacked down. Scleroderma affects the motions of the joints, and when it occurs about the chest and throat may interfere with respiration. It appears to be a condensation of the fibrous layers of the skin, so that the bundles of connective-tissue fibres are packed closely together and are increased in number. It is chronic, is not very dangerous, and is best treated by massage and lubricating applications.

ACUTE CIRCUMSCRIBED OR ANGIO-NEUROTIC ŒDEMA.

A lesion of the skin which has been termed *acute circumscribed œdema* is represented by the sudden appearance of circumscribed swellings of certain parts of the body, varying in intensity and size in different localities. It is closely allied to urticaria, and was formerly described under the name of *giant urticaria*. We do not know much about either its cause or its pathology. I have sometimes met with it in children in whom it was evidently of reflex origin, depending, probably, upon irritation in various parts of the body, such as the mouth, the genitals, and the gastro-enteric tract.

It is not dangerous, may occur at any age, and its treatment is simply symptomatic.

The following cases illustrate the disease:

A little boy, two and one-half years old, had had diarrhœa during the summer, and had been left in rather a weak, debilitated condition. He had for some weeks been pale, fretful, and constipated. His appetite had been capricious, and he had not cared to take any food but milk. When he was nineteen months old an egg had been given to him, which he vomited, and later a slight swelling of both eyes had occurred, lasting for a day or two.

When I saw the child the history that was given me was that in the morning he had eaten an egg. Soon after he became rather dull and cross, but did not vomit. A slight swelling of both eyes was then noticed, and later, when I saw him, the right eye was very much swollen, so that the conjunctiva was corrugated, and the tissues of the eyelids and of the cheek under the eye were so swollen that the eye itself could be examined only with the greatest difficulty. Each time that the child had eaten an egg this swelling occurred in about fifteen minutes. In the course of a few hours the swelling passed off, and did not return. An examination of the urine showed nothing abnormal.

Another instance of this kind occurred in a boy of three years, in whom the peripheral irritation was evidently dependent upon a tight and irritating prepuce. In this case sudden œdematous swellings of the fingers and backs of the hands would occur at irregular intervals, lasting for a few hours, and would then entirely disappear. These manifestations continued until the child was circumcised, since which time the symptoms have not returned. In this case, also, the urine was found to be normal.

DIVISION VIII.

SPECIFIC INFECTIOUS DISEASES.

The number of infectious diseases which are supposed to be caused by some specific infection is so rapidly increasing that at present no definite classification of them can be made. In like manner our knowledge of the specific organism which in an individual case produces the disease is so continually advancing that we can only make a provisional division into somewhat arbitrary groups of the diseases which we speak of as infectious. Thus, in certain diseases, tuberculosis, cerebro-spinal meningitis, typhoid fever, diphtheria, epidemic influenza, malaria, tetanus neonatorum, erysipelas, certain forms of ileo-colitis, and cholera asiatica, the micro-organism is known; while in others, such as epidemic parotitis, pertussis, syphilis, and acute articular rheumatism, which are evidently just as infectious, the specific micro-organism has not yet been discovered. Again, there are certain infectious diseases which have usually been classed in a group by themselves on account of the marked similarity which they present in their general characteristics. This group, as a whole, has been designated as the exanthemata, and comprises scarlet fever, measles, rubella, variola, and varicella.

TUBERCULOSIS.

Tuberculosis is an infectious disease due to the invasion of the tissues by the tubercle bacillus.

General Etiology.—Intra-uterine infection of the fœtus by a tuberculous mother from direct infection may occur. Infection by the father from the mother, or from the ovum of the mother has not been proved. In the great majority of cases the child is infected after birth. This infection may be by direct inoculation from the mouth, as in the Jewish rite of circumcision, or, as is the most common means of transmission, from the inhalation of the bacilli contained in dry sputum. A less common mode of invasion is that from uncooked food. The bacillus tuberculosis may be present in cow's milk, and very exceptionally in the milk of human beings.

The question of the relation of bovine tuberculosis to human tuberculosis and the danger of infecting man from the milk of tuberculous cows have received renewed attention of late. The researches of several investigators, particularly those of Smith, seem to show that the bovine tubercle bacillus is more virulent for certain species of animals than the human tubercle bacillus. Whether the two organisms are distinct or whether the differences which have been noticed between them are simply modifications brought about by the adaptations of the bovine bacillus to the different conditions in the human body are points still to be determined. The weight of evidence, however, in regard to infection from milk is quite sufficient to justify the following practical conclusions:

(1) That tuberculosis in many cases may be transmitted to animals either through the ingestion or inoculation of the milk of cows that are tuberculous. (2) That the milk of such cows may be infectious even when there is no tubercular disease of the udders. (3) That, therefore, the milk of certain tuberculous cows contains living, virulent tubercle bacilli. (4) That, whereas, from the nature of the case, it is impossible by direct experimentation to prove that the milk of certain tuberculous cows is pathogenic for man, nevertheless the clinical evidence of competent observers, strengthened by the results of animal experimentation, makes it practically certain that tuberculosis may be produced in man by the ingestion of the milk of infected cows. (5) That it is, therefore, of the utmost importance that the milk supply, especially that which is used as food for infants and young children, shall be from an absolutely reliable source.

The infection of food by flies carrying the bacilli from one point to another is possible. Direct infection may also take place by the skin, and in these cases it produces a local lesion. The bacilli may pass through a mucous membrane without infecting it, and may be taken up by the lymphatics and carried to the lymph-nodes where their progress is usually arrested for some time.

Tuberculosis is a very prevalent affection in early life. It is very rare in the new-born and infrequent in the first three months of life. After this age the number of cases increases rapidly, and the disease is very common in the latter part of the first year and in the second year, gradually growing less common as puberty is approached. The presence of tuberculosis in children is very much underestimated. In two hundred and twenty autopsies of diphtheria, tubercular lesions were found usually in the lungs, and sometimes in the lymph-nodes of the mesentery, in sixteen cases. The disease is very much more common in young children than has been supposed, and it may exist before there are any symptoms at all.

The bacilli find entrance into the body in various ways. The chief mode of entrance is probably through the respiratory tract. The organisms may also enter through the alimentary canal or by means of the skin. As a rule, where the bacillus finds an entrance into the tissue it produces at that point the characteristic lesion of the disease. In other cases close examination fails to reveal any lesions at the point of entry, although this does not preclude the possibility that the lesions may be present. It is probable that the bacilli enter into the circulation in small numbers, and are conveyed to various tissues of the body, as the bones or joints, and the disease may appear as a primary infection in organs remote from the point of entrance. Frequently the bacilli, without apparently producing any lesions at the point of entry, enter into the lymphatics and are carried to the lymphatic glands in which the disease first appears. Having gained an entry into the tissue the disease always extends by means of the bacilli, which are carried from place to place through the natural canals or channels of the lymphatics which are in proximity to the diseased focus, or by means of the blood. In all cases of tuberculosis it is probable that there is some infection of the blood. A few bacilli find their way into the blood, and are deposited by it in those organs in which the circulation is most favorable for their arrest. We have an example of this in the almost constant miliary tuberculosis of the liver when the infection is elsewhere. At times the bacilli find their way into the blood in large numbers. This general infection of the blood is due either to a tuberculosis of the blood-vessels or to a tuberculosis of the thoracic duct. The bacilli, entering into the blood in large numbers, are conveyed into the different organs of the body and give rise to the condition known as *acute miliary tuberculosis.*

PREDISPOSITION.—The predisposition to tuberculosis may be hereditary or acquired. It is hereditary in the sense that the individual inherits tissues which are more or less receptive to, and which provide a favorable material for the development of the tubercle bacillus. In diseases resulting in a great reduction of the vitality, and in certain acute infectious diseases, such as measles, pertussis, and epidemic influenza, a tuberculous predisposition seems to be acquired. Bad food and poor hygienic surroundings, especially absence of sunlight in the crowded tenement districts of large cities, render the tissues more vulnerable to the tubercle bacillus. In like manner any debilitating disease, by lowering the resistance of the tissues, acts as a predisposing cause. Repeated attacks of catarrhal inflammation of the mucous membrane of the nose, throat, bronchi, or intestinal tract render these parts more vulnerable. Infectious diseases, especially those in which the respiratory organs are affected, predispose to tuberculosis. In these cases it would seem that the bacilli have been latent in the lymph-nodes surrounding the bronchi until, under the irritation of the new disease, they become active again.

GENERAL PATHOLOGY.—Tuberculosis is characterized by the production of tissue in which there is proliferation and an inflammatory exudation. The proliferation of tissue leads to the formation of epithelioid and giant cells. The inflammatory exudation may be serous, fibrinous, or purulent. Usually in the tubercular lesions there is both the tissue proliferation and the inflammatory exudation. The proliferation of tissue occurs in the form of small nodules which are called *miliary tubercles*. The tuberculous tissue is further characterized by a form of necrosis, and the necrotic tissue has a tendency to soften and break down. These miliary tubercles often coalesce so as to form what is known as *conglomerate tubercles*, in which the process of necrosis is especially marked, giving rise to the "cheesy" nodules so characteristic of the disease. Certain degenerative changes are common in tuberculosis. Thus fatty degeneration frequently occurs from the diminished oxygenation due to the destruction of lung tissue. Again, amyloid degeneration is common in those forms of tuberculosis which run a chronic course, and in which there is marked destruction and suppuration of tissue. The lesions of tuberculosis are very numerous, and in the child do not differ materially from those which are found in the adult. The ordinary chronic tubercular lesions met with in the adult are seldom seen in children.

The younger the child the more likely is tuberculosis to be chiefly located in the lung. As the child grows older the meninges are commonly affected, and later the peritoneum, intestines, and joints. During the first two years of life the lesions of meningeal tuberculosis are rare in comparison with the period beginning with the third year, and the pulmonary lesions usually result fatally before there has been extensive invasion of the intestines and joints. As the child passes into its third year the marked pulmonary lesions lessen in frequency and the meningeal forms become more common. Later the joints become a prominent nidus for meningeal infection, and in older children the peritoneum, intestines, and mesenteric lymph-nodes show, more commonly, marked lesions. At all ages, however, there are usually found pulmonary lesions more or less marked whenever infection has taken place with the tubercle bacillus.

PROPHYLAXIS.—In considering the question of prophylaxis in tuberculosis, special attention should be paid to what has already been said of the modes of invasion and of predisposition. In all cases in which there is a family history of tuberculosis the child should, if possible, be surrounded with unusual precautions; a change from a severe to a mild climate, especial care of even mild catarrhal affections of the mucous membranes, much fresh air, but dry, warm, fresh air, protection from inhalation of dust, rooms with a sunny exposure, and freedom from living in the house with a tuberculous individual, are precautions especially to be observed. Exposure to the diseases which have been mentioned as most likely to be followed by tuberculosis should be carefully avoided. Tuberculous nurses should never be allowed to take care of children, tuberculous individuals should not be allowed to kiss children, and the sputum from a tuberculous individual should be destroyed at once before it has become dried. A tuberculous mother or wet-nurse should not be allowed to nurse an infant. The animals which provide milk should, at stated intervals, be carefully tested with tuberculin, and when the source of the milk is not known or doubtful, the milk should be heated to 68.3° C. (155° F.) for half an hour.

The following case, seen by me in consultation with Dr. W. L. Richardson and Dr. H. P. Jacques, illustrates the importance of prophylaxis:

A boy, five years old, died of tubercular meningitis. The autopsy showed extensive tubercular lesions of the meninges with enlarged bronchial lymph-glands and cheesy nodules at the apices of both lungs. The child up to the time of the attack had always been perfectly well. There was no history of tuberculosis in the family. There were several other children, none of whom had ever shown any symptoms connected with tuberculosis. This boy, at the age of fourteen months, was placed in the charge of a nurse, who remained with him until he was four and a half years old. Just before leaving the child, she was brought into especially close connection with him while his parents were away for some weeks. The child was very fond of her, insisted on being in her lap a great deal, kissed her on the mouth, slept in her bed, and kept her in the nursery with him continuously. The nurse, while taking care of the child, developed tuberculosis of the lungs and subsequently died of this disease. Other cases of this kind have been known to occur.

General Symptomatology.—After a variable period of loss of weight and general health, which especially occurs in cases in which acute miliary tuberculosis is secondary to measles or to pertussis, the infant begins to have an irregular type of fever, cough, and general symptoms, such as diarrhœa, capricious appetite, anæmia, and change of temperament. In some cases the disease appears with a sudden rise of temperature and advances very rapidly, but it is often of a subacute type, and frequently, unless the tuberculosis markedly affects some organ, such as the lung, the symptoms are very obscure, death finally taking place from exhaustion or from the development of some localized condition, such as tubercular meningitis or some acute pulmonary complication, bronchopneumonia being especially common.

General Diagnosis.—From what has been said concerning the symptoms of tuberculosis, it is evident that when no localized tubercular lesions are found, the diagnosis, excepting by supposition, is not possible. It is probable, however, that when more is known regarding the use of tuberculin in infants and in young children, this method of diagnosis will be more frequently used, and will prove of value by providing us with a safe method for detecting an incipient tuberculosis, and thus aid us in preventing the development of later and more serious lesions.

Tuberculin Test.—The technique of the tuberculin test as described by Koch is as follows: One c.c. of tuberculin added to 999 c.c. of distilled water equals $\frac{1}{1000}$, and 1 c.c. of this solution equals 1 milligramme. The dose of this solution for an infant or young child is from ½ to 1 c.c. In using this test a record of the child's temperature must first be kept every two hours for twenty-four hours. If it is then found that the temperature is irregular or high the test is not of much value. If, however, the temperature is regular and not over 38.3° C. (101° F.), the test can be used, and has been found in my experience at the Infants' Hospital to be of considerable value. After the part of the skin to be injected has been thoroughly washed first with soap and water, and then with alcohol, and the hands of the operator and the syringe disinfected, from ½ to 1 c.c. of the solution are injected into the arm or leg. If the child is not tubercular no symptoms will be noticed. If it is tubercular the temperature will rise in from eight to twenty-four hours after the injection, and will fall again within about twenty-four hours. During the period of reaction there may be a certain amount of malaise, but in my experience there is little or no danger of serious results.

General Prognosis.—The prognosis of tuberculosis in early life is almost invariably bad. Even when the bacilli have been encapsulated they are liable at any time to become active.

General Treatment.—Of especial importance in the treatment of children with tuberculosis in any form is the general hygiene, fresh, dry air, a warm, dry climate, and plenty of sunshine. The treatment by drugs is very unsatisfactory and usually without good results. In some cases cod-liver oil seems to be of temporary benefit. Cream is useful and should be given when cod-liver oil is not well borne or is especially distasteful. A general diet adapted to the age and condition of the especial child, and the treatment of symptoms which arise when special organs are involved, are the only rational procedures. Tuberculin has not as yet been proved to be sufficiently valuable as a therapeutic agent to be accepted by the medical profession in general.

The following case illustrates how extremely latent and masked may be the symptoms of tuberculosis

An infant, seven months old, was in the Infants' Hospital from October until December. During this time it became extremely emaciated, diarrhœa occurred from time to time, and there was an irregular and varying temperature, never especially high. It had a purulent discharge from the right ear, and a serous discharge from the left ear. There were no other symptoms and no abnormal physical signs. It failed rapidly and died. The autopsy showed miliary tuberculosis of the pleura, spleen, kidney, and liver, chronic tuberculosis of the bronchial lymph-glands and of the lung, and broncho-pneumonia.

GENERAL TUBERCULOSIS.—General tuberculosis in early life may be *acute* or *chronic*. It is now believed that in every case there is a tubercular focus, usually in a gland or in the lung, from which the general infection has emanated. The early symptoms of a general tubercular invasion vary much and are often obscure. As a rule, they appear before the symptoms of local tuberculosis of an especial organ, and are represented by irregular temperature, anæmia, and a general lowering of the nutrition and vitality.

ACUTE MILIARY TUBERCULOSIS.

The term *acute* is somewhat misleading in speaking of miliary tuberculosis, since this form of general tuberculosis varies greatly in the rapidity of its invasion and in the intensity of its symptoms, according to the rapidity with which the bacilli gain an entrance to the circulation, and in proportion to the susceptibility of the individual to their virulence. Some of these cases are extremely acute, while others are markedly chronic. There are certain organs of the body which are rarely affected. This must be attributed to the fact that the organs in question do not offer favorable conditions for the development of the bacillus. Thus, it is extremely common for the bronchial lymph-nodes and the lungs to be affected, while it is rare for the stomach or the genito-urinary organs to be markedly involved. Especially noticeable in comparison with those acute cases which occur usually in somewhat older children with pronounced symptoms, and representing the form which is called the *typhoidal* type of the disease, is the clinical picture represented by those cases which occur in early infancy, and which are markedly subacute rather than acute, simulating closely *infantile atrophy*.

PATHOLOGY.—The disease is characterized by the formation of nodules, varying in size and character, in the different organs and tissues of the body. As a rule, the nodules (miliary tubercles) are larger and not so characteristic as in the adult. As compared with the adult, they vary somewhat in their distribution. They are much more numerous and larger in the liver in the case of the child than they are in the adult. The condition is produced by the entry of the tubercle bacillus in large numbers into the blood. They may enter the blood through a blood-vessel, or by means of infection of the thoracic duct following infection by the lymph-nodes. It is not probable that they multiply directly in the circulating blood, but they may multiply in the blood-vessels of organs in which the blood-stream is comparatively inactive, as in the liver.

(1) **Acute Miliary Tuberculosis Simulating Typhoid Fever.**—SYMPTOMS.—The symptoms of this type of the disease are very indefinite. There is rapidly progressive emaciation. The temperature is irregular, and fluctuates from 37.2° or 37.7° C. to 40° or 40.5° C. (99° or 100° F. to 104° or 105° F.), or even higher. The respirations are often accelerated beyond what can be explained by the fever, and physical signs are markedly absent in the lungs. The pulse is rapid. Certain cases of this class simulate typhoid fever by presenting symptoms of apathy, headache, slightly enlarged spleen, and tympanites. In making the differential diagnosis from typhoid fever it is well to take into consideration the family history in regard to tuberculosis, and also whether the child has been living where typhoid was prevalent. Although no leucocytosis is present in either disease, yet it has been held that while in typhoid there is a relative increase in the small mononuclear cells, there is in tuberculosis a relative decrease. In other respects

the blood simply shows the characteristics of a secondary anæmia. The *Widal reaction* is absent in tuberculosis. The tubercle bacillus may or may not be present in the sputum. In some cases the bacilli of typhoid and tuberculosis have been found in the urine. The tuberculin test is seldom of value in these cases, as the fever is continuous and usually sufficiently high to prevent the characteristic rise of temperature from being perceptible. The less regular temperature, the rapid respirations, and the absence of rose spots are significant of tuberculosis. Finally, in most cases the tendency of typhoid fever is gradually to recover, while tuberculosis shows progressive emaciation and the development of new symptoms according as other organs are involved. In some cases malaria may simulate tuberculosis, but the presence of the plasmodium as determined by a blood examination and the response to the treatment with quinine serve to separate this disease from tuberculosis.

Prognosis.—The course of this type of the disease is short and the result invariably fatal.

(2) **Acute Miliary Tuberculosis Simulating Infantile Atrophy.**—Symptoms.—I have frequently had patients brought into the Infants' Hospital in whom it was impossible to differentiate in the beginning, and perhaps for weeks, what eventually proved to be miliary tuberculosis. The symptoms in these cases are simply progressive emaciation, with occasionally a temperature moderately raised, but in no way differing from what is frequently found in the atrophic condition in which a slight disturbance of digestion may cause a similar rise in temperature. These tubercular cases simply die of exhaustion, with no physical signs developing during life, the disease being completely masked and only recognized at the autopsy. In some cases, however, after a variable period, physical signs can be detected in the lungs, the temperature rises more and more, and there may be cough and accelerated respirations. In these cases, also, there are at times diarrhœa and general gastro-enteric disturbance, but these conditions depend entirely on the reduced condition of the infant and not upon tubercular lesions of the intestine. The temperature in these cases varies from 37.2° to 38.8° C. (99° to 102° F.). It must be remembered that infants with simple infantile atrophy at times develop non-tubercular broncho-pneumonia and die of it, so that a diagnosis made by finding physical signs in the lungs in those cases which simulate infantile atrophy is not conclusive, as the signs do not necessarily prove that tuberculosis is present. On the other hand, acute miliary tuberculosis of the lung usually presents no physical signs whatever, so that the differential diagnosis between many cases of acute miliary tuberculosis and infantile atrophy must be held in abeyance, and, unless the tuberculin test can be used, cannot be made except at the autopsy.

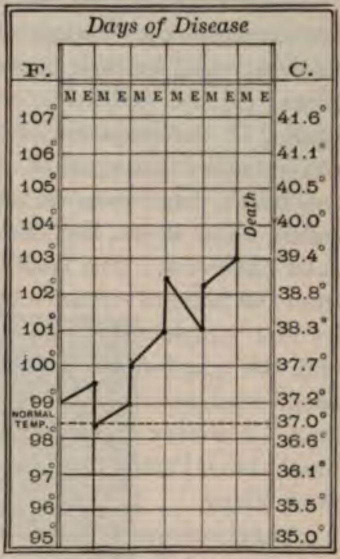
Prognosis.—The prognosis is fatal.

Treatment.—The general treatment of acute miliary tuberculosis is essentially symptomatic and by the use of stimulants. When the disease lates typhoid fever, the treatment should be such as is described in disease, on the supposition that it may turn out to be typhoid. When nulates infantile atrophy, however, the ment should be the same as in that se, and is essentially dietetic.

'he following case was one of acute ry tuberculosis:

n infant, one and a half years old, was it to the Infants' Hospital to be treated for ack of bronchitis. On entrance it was much ated and failed rapidly. No marked signs l a subacute bronchitis were found. There times a slight cough. The temperature was ately raised and of an irregular type. The and legs, especially the buttocks, showed ous subcutaneous abscesses, and there were few on the head. Five days before the inied the temperature rose as represented on 4. The post-mortem examination showed iere was chronic tuberculosis of the bronlands, with acute miliary tuberculosis of the lungs, spleen, kidneys, liver, and meninges.

CHART 4.



Acute miliary tuberculosis.

CHRONIC GENERAL TUBERCULOSIS.

addition to the lesions which characterize *acute* miliary tuberculosis, *nic general* form of tuberculosis occurs in certain cases. The symp- are more marked than in the acute form, and usually are so closely ected with the bronchial lymph-nodes and the lungs that it is more diagnosticated. This chronic general tuberculosis is one of the common forms of tuberculosis in children.

OCALIZED TUBERCULOSIS.—Although tuberculosis may by a al infection invade all the tissues, thus constituting the general tuberis just described, it is apt to be so pronounced in certain parts, and ich more common in some than in others, that these localized forms est be described separately. An instance illustrating this is given in ymph-nodes of the neck in children where the node is very comy found to be tuberculous, and yet in many cases no trace of a genuberculosis can be found elsewhere.

TUBERCULOSIS OF THE LYMPH-NODES.

uberculosis of the lymph-nodes is an exceedingly common form of culosis in children. In all forms of tuberculosis the lymph-nodes are ed. The tubercle bacilli come in very close relation with the lymph s and ducts and are carried by them into the lymph-nodes belonging part. There are also cases of tuberculosis of the lymph-nodes in which the disease appears to be primary in these parts. Such cases are found in the lymph-nodes of the neck, of the axillæ, and of the mesentery.

Tuberculosis of the lymph-nodes appears in two forms: (1) the nodes are enlarged and contain the tubercular tissue in the form of *miliary tubercle*, which by their increase and coalescence can form large caseous areas; (2) the nodes are also enlarged in the form of *diffuse tubercles*, leading to the caseation of large areas, in fact, the entire node may be converted in consequence of this caseation of the diffuse tubercular tissue into a large caseous mass. The caseous tissue of the nodes may soften, break down, and even suppurate, leading to the formation of tubercular abscesses. The bacilli having entered at some point can be carried from the point of entrance by the lymph-streams into the nearest lymphatics and lymph-nodes, without the production of local lesions. In general, the lymph-nodes play an important part by preventing the tubercle bacilli from entering into the blood, and the disease from extending further. It is very interesting to note that the lymph-nodes can soften and break down in this way and be discharged, and that no further infection need follow.

Tuberculosis of the Cervical Lymph-Nodes (Scrofula).—The most important of the lymph-nodes affected in tuberculosis are the lymph-nodes of the neck. Invasion of the cervical lymph-nodes may occur primarily by infection from the throat. When this invasion occurs in connection with tuberculosis of the lungs the infection of the cervical nodes is probably primary to the pulmonary lesions. The cervical lymph-nodes are more commonly affected by the tubercle bacillus in the early period of childhood than in infancy, in which the infection of the bronchial nodes are so frequently met with. When the term scrofula was in more general use, tuberculosis of the cervical lymph-nodes was considered as one of its most frequent manifestations; now the term can be dropped entirely.

SYMPTOMS AND DIAGNOSIS.—In cases in which the cervical lymph-nodes begin to enlarge in a marked tuberculosis, as of the lung, the symptoms are those of a slow process extending over months, with a tendency to suppuration.

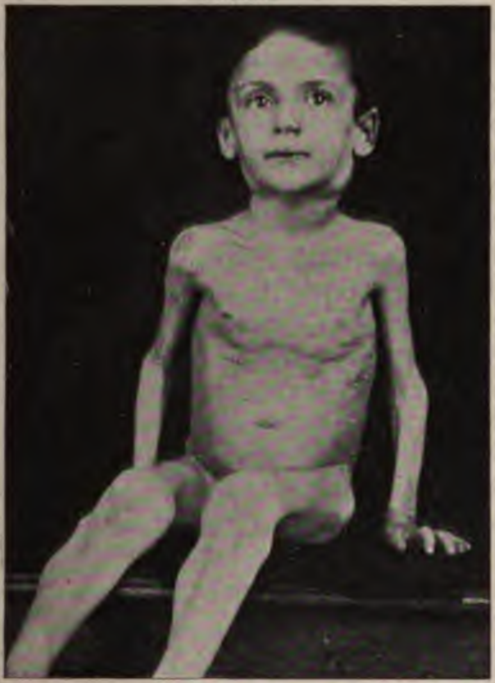
The diagnosis in these secondary cases is made by eliminating the causes of glandular enlargement which could produce a similar hyperplasia by reflex action, or which could be produced by some other organism. The diagnosis, however, is chiefly made by the tubercular history of the case, as in the majority of cases the nodes under these circumstances are tubercular.

Primary tuberculosis of the cervical lymph-nodes is to be distinguished from other forms of infection by its slow progress, by its greater tendency to suppuration, and also by the fact that the suppuration as a whole takes place later than the simple form. The enlarged cervical nodes due to syphilis, Hodgkin's disease, or malignant growths should be differentiated from tuberculosis of the cervical lymph-nodes, as described in these diseases.

PROGNOSIS.—The prognosis of tuberculosis of the cervical lymph-nodes is good, the exception being for the child to develop tubercular disease elsewhere. The prognosis when the tuberculosis is secondary to tuberculosis elsewhere varies according to the tubercular lesions from which it originates.

TREATMENT.—When the tubercular disease has extended to other organs there is seldom much benefit to be derived from the treatment of these nodes. Although it is conceded that general tuberculosis following tubercular cervical adenitis is exceedingly rare, yet there have been cases reported in which such infection has taken place, and the glands should be removed unless they have grown smaller in a number of months, at any rate if they show signs of softening. A change of climate is indicated, and if the child has a tubercular inheritance especial care should be taken to keep the naso-pharynx and throat in a healthy condition. Adenoid growths and enlarged tonsils should be removed. Any lesion of the face and head which may cause irritation should be treated, as, by their reflex action, they may cause increased activity of the tubercle bacilli.

FIG. 99.



Chronic pulmonary tuberculosis, with involvement of the cervical lymph-nodes.

Medicines, except for special conditions, such as anæmia, are useless.

External applications of an irritating nature are contra-indicated. Preventing the head from rolling from side to side, and thus pressing on the inflamed glands, can sometimes be accomplished by various mechanical devices in the shape of collars. The operative treatment includes not only the removal of the gland, but the removal of it at such a time that the smallest scar will be left, as in many cases following a suppuration of these glands unsightly scars are left which disfigure the child for life. These cases should, therefore, be placed in the hands of a skilful surgeon.

Fig. 99 represents a child with tuberculosis of the cervical lymph-nodes in a case of marked chronic tuberculosis of the lungs.

Tuberculosis of the Bronchial Lymph-Nodes.—Of all the lymph-nodes in the body, the bronchial lymph-nodes are probably the most frequently infected, due to the fact that the primary focus of infection is so commonly found in the lungs, and, as the lymph flows from the lung towards the nodes, the nodes may be infected in this way by the lung. Thus, the bronchial lymph-nodes protect the body from general infection, but do not protect the lungs, and such tubercular lymph-nodes can become, moreover, a starting-point for further extension of the disease after the primary lesions by which the lymph-nodes themselves became infected have been recovered from.

Tuberculosis of the bronchial lymph-nodes, if it is extensive, may involve the pneumogastric nerve. The nodes may possibly suppurate into a bronchus, and the infection can in this way also extend into the lungs.

SYMPTOMS.—Various symptoms may arise from the breaking down and ulceration of the bronchial lymph-nodes, or by their pressure they may cause irritation of the neighboring parts. Thus, an annoying cough may be produced by local irritation. Loeb has reported cases in which the previous symptoms had been only cough, and in which an attack of suffocation caused death. Adelbert and Vogel have reported cases in which ulceration originating from these points has resulted in death. The physical signs are indefinite and unreliable, and we must depend upon the general symptoms of spasmodic cough and dyspnœa, without an adequate explanation of these symptoms being found elsewhere.

Tuberculosis of the Mesenteric Lymph-Nodes.—Tuberculosis of the mesenteric lymph-nodes may be seen without any evidence of tuberculosis elsewhere in the body, or it may be found in connection with intestinal tuberculosis. In all cases the infection is by means of the intestine, although there may be no lesions at the point of invasion.

When the nodes can be reached or definitely localized, they should be removed, even if laparotomy has to be performed; for there is always danger not only of their breaking down and becoming a source of general tubercular infection, but also a peritonitis may arise suddenly at any time from acute inflammation of the peritoneum originating from a disintegrated node and ending fatally in perhaps a few days. A case of this kind is described on page 843.

TUBERCULOSIS OF THE LARYNX AND TRACHEA.

Tuberculosis of the larynx and trachea is very rare in infants and rare in young children. When it is present it is invariably secondary to tuberculosis of the lungs. The symptoms are the same as those of non-tubercular disease of these organs, and the diagnosis can only be made definitely by finding the tubercle bacillus in the sputum.

Treatment.—The treatment does not differ from that of the non-tubercular cases.

TUBERCULOSIS OF THE LUNGS.—The tubercle bacillus may be brought to the lungs by means of the blood or by the respiratory passages, the latter, according to Northrup, being the most frequent in children, the bacillus entering with the inspired air and lodging in the mucous lining of the bronchi or in the alveoli. When carried by the blood they are generally scattered throughout the tissue and give rise to miliary tubercles. In some cases the infection may arise from the throat with or without the production of a local lesion, the bacilli being carried through the lymph-nodes into the blood and from the right side of the heart to the lung. Rarely a very few bacilli may be brought to the lungs by the blood. They may affect only a certain definite portion of the lung and give rise to lesions which are scarcely distinguishable from the lesions produced by the entry of the bacilli through the respiratory passages. Tuberculosis of the lungs may occur in three forms: (1) *acute miliary tuberculosis*, (2) the more diffuse *tubercular pneumonia*, and (3) *chronic tuberculosis* of the lungs.

Ordinarily the distinction made between acute miliary tuberculosis and tubercular pneumonia lies in the preponderance of the exudative lesions in the latter. In the lungs of children there is a much greater tendency to exudation than in adults, and even in acute miliary tuberculosis the essential proliferative changes are, as a rule, accompanied by exudation. There is a further difference between children and adults in that the chronic forms of tuberculosis which are accompanied by excessive destruction of tissue, with inflammation leading to a formation of fibrous tissue, and producing the condition known as fibroiditis, are seen to a much less degree in children than in adults.

(1) ACUTE MILIARY TUBERCULOSIS OF THE LUNG.

Acute miliary tuberculosis of the lung has been described on page 387 as a part of acute general miliary tuberculosis. It presents no other symptoms, when the acute process is limited to the lung, than those which have been already mentioned as occurring in the acute general miliary tuberculosis. There may, however, be a formation of miliary tubercles in the lung in combination with other forms of tuberculosis of this organ, the formation of tubercles being a more chronic process than in the acute disease.

(2) ACUTE TUBERCULAR BRONCHO-PNEUMONIA.

Etiology.—Acute tubercular broncho-pneumonia is common in children from the sixth month to the fifth year, a large proportion of the cases, however, occurring after the second year. It may begin in the lungs, or may be secondary to any other of the lesions of general tuberculosis, or it may follow any non-tubercular lesion of the lungs, such as bronchitis or broncho-pneumonia. It is common in children who have been debilitated by previous illness, and occurs especially after measles, pertussis, scarlet fever, and diphtheria, being most frequent in the first two. It may, however, develop in perfectly healthy, well-nourished children, and, as Osler has expressed it, may be a terminal process in cases in which a local tubercular disease exists in other parts, such as the skin, bones, lymph-nodes, or the uro-genital tract.

Pathology.—The infection extends by means of the bronchi, and the lesions extend through the bronchi and the alveoli into the surrounding tissue. This is a true pneumonia. There is an exudation into the alveoli of the lungs, which consists chiefly of fibrin and leucocytes. In addition to this, there are numbers of large cells, which probably arise from proliferation of the epithelium lining the alveoli. Both the exudative and the proliferative cells undergo caseation, and the tubercular pneumonia may occur in the form of discreet nodules similar to ordinary forms of broncho-pneumonia, or in the form of larger areas, which may arise either by confluence of the smaller foci or by the simultaneous affection of a large area of the tissue.

As in the other forms of broncho-pneumonia, the initial lesion is a bronchitis and peri-bronchitis, the distinguishing tubercular features being caseation and necrosis of the consolidation with the presence of the tubercle bacilli. The accompanying phenomena of atelectasis and emphysema occur as they do in non-tubercular broncho-pneumonia. In some cases the non-tubercular broncho-pneumonia precedes the tubercular disease, this occurring particularly after measles, scarlet fever, diphtheria, and pertussis. When the tubercular broncho-pneumonia follows the non-tubercular form, in addition to the lesions of the latter disease, there are found true tubercular processes, such as peri-bronchial nodules, tubercular infiltration, and caseous areas.

It is rare for the tubercular process in children to begin at the apices of the lungs and gradually extend downward, as is common in adults. When this occurs it is usually in the later years of childhood, when the conditions are beginning to approximate those of later life.

Tubercular broncho-pneumonia may be acute or chronic, but it is rarely very chronic. It may occur in very small foci apparently following the bronchi, or it may invade a much larger area and even an entire lobe of the lung.

Symptoms.—The symptoms of acute tubercular broncho-pneumonia are very similar to those of non-tubercular broncho-pneumonia. According to Osler, in most cases the onset of the disease simulates that of the ordinary non-tubercular broncho-pneumonia so closely that a differential diagnosis between the two diseases cannot be made until after death, and even then the post-mortem appearances may not be those distinctive of tubercular disease, and the pathological diagnosis can be determined only by finding the tubercle bacillus. Children may be attacked with cough, a heightened temperature, and the physical signs of broncho-pneumonia. These signs, as would naturally be expected, are usually found in the back and lower portion of the lung rather than at the apices, as in adults, on account of the usual nidus of the tubercular lesions,—namely, the bronchial lymph-nodes. In some cases the onset of the disease is not so acute, and its course not so rapid. The child emaciates and has only a moderate temperature, but later the development of such symptoms as sweating, chills, and hectic fever, together with the signs of softening and breaking down of the lung-tissue, leads us to suspect that we are dealing with tuberculosis of the lung.

Diagnosis.—The diagnosis, as a rule, is to be made by taking into consideration the family history of the child, as the tissues of children whose parents are tubercular show an especial liability to infection by the tubercle bacillus. Careful investigation should be made as to whether the child has been exposed to tuberculosis in any form; whether it has itself shown signs of any localized form of tuberculosis; and especially whether it has recently had an attack of measles or pertussis. The physical signs are in no way characteristic, as they may not only be similar to those of the non-tubercular form of broncho-pneumonia, but also we must remember that the various local conditions of chronic interstitial pneumonia and persistent broncho-pneumonia, although simulating tubercular disease, may in children be non-tubercular, Continuous fever of a rather high grade in comparison with the non-tubercular pneumonia, the greater intensity of the anæmia, and emaciation mark the tubercular invasion. When the symptoms develop insidiously, especially following measles and pertussis, instead of beginning during the course of these diseases, tuberculosis is probable. It must, however, be remembered that simple non-tubercular processes may last for many months, and the children finally recover. The diagnosis can be made positively only in those cases in which a specimen of the sputum can be obtained and examined for the tubercle bacillus.

Prognosis.—The prognosis is invariably unfavorable.

Treatment.—The treatment of tubercular broncho-pneumonia is the same as that of the non-tubercular forms (page 701).

(3) CHRONIC TUBERCULOSIS OF THE LUNGS.

Chronic Diffuse Tuberculosis.—Chronic tuberculosis of the lungs as it is ordinarily met with in adults is rarely seen in young children.

During the first three months of life tubercular disease of any form is very rare, but in the latter part of the first year it becomes very common. The tubercular lesions which are found in the lungs in later life also occur in early life. Although cavities are not so commonly found in young children as in adults, it is not so much that they do not exist, but because they are located at the root and central portions of the lung, and are, therefore, more difficult to detect on physical examination. It has been noticed that large cavities at the apex of the lung are rare in early life, but become more common as the child grows older. Tubercular disease of the lung is very irregular in the extension of its lesions in young children. Much more advanced lesions are usually found at the post-mortem examination than are detected during life. The primary lesion of chronic tuberculosis of the lungs is commonly a tubercular broncho-pneumonia.

Chronic Localized Tuberculosis.—In this form, from a single tubercular focus, there is an extension of the disease by continuity. The bacilli find their way into the surrounding lung-tissue by means of the lymphatics, or by means of the infection of the adjoining alveoli. Nodules varying in size may be produced. In this form of localized tuberculosis there seems to be a high resistance of the tissue to the tubercle bacillus, and there is not the same tendency to an extension of the infection as in the other forms. After undergoing caseation the tissues seldom remain in this condition, for the caseous material tends to soften, and is discharged by means of the bronchi, or it may be partly absorbed, leaving a cavity in its place.

SYMPTOMS.—The symptoms of chronic tuberculosis of the lungs differ but little in the child from those seen in the adult, and are marked by the same irregularities in their course. This is due to the varied forms of the lesions. In young infants the symptoms are so often obscure and the physical signs of the serious pathological conditions which exist in the lungs are so frequently masked that the diagnosis is apt to be very doubtful. There is often a history of tuberculosis in the parents. The more common symptoms of chronic tuberculosis of the lungs are gradual loss in weight, strength, and appetite, irregular and moderate hectic fever, and sweating. The physical signs are slowly increasing dulness in certain areas of the lung, especially in the back, accompanied by râles and the other signs of solidification. Later in the disease the characteristic signs of cavities may develop. Cough is usually present, although it is sometimes so slight in the beginning as not to be especially noticed by the parents. Hæmoptysis is rare in infants and in young children, but may be present in older children as they approach the age of puberty. As the disease progresses there is dyspnœa, usually of a moderate grade, with cyanosis, but in some cases considerable destruction may have taken place in the lung-tissue without the presence of any especial dyspnœa.

The course of chronic tuberculosis of the lungs is rather more rapid in children than in adults, and it is seldom that the long-protracted course of the disease so frequent in adults is met with in children. Sometimes, however, the child improves in its general health and may live for many years. In these cases the terminal phalanges of the fingers may become clubbed, and there is usually dyspnœa on exertion.

DIAGNOSIS.—The diagnosis is to be made from chronic empyema and from chronic non-tubercular broncho-pneumonia. The former disease can be readily eliminated by making an exploratory aspiration, but the latter can often be distinguished only by means of a bacteriological examination. In older children, from whom a specimen of the sputum can be obtained, the diagnosis is readily made by the detection of the tubercle bacillus. In younger children, in whom expectoration does not take place, the diagnosis is much more difficult, but if the children are carefully watched it is often possible to obtain a specimen of the sputum if the child happens to vomit, in which case particles of sputum may be coughed up with the vomitus and can be separated from it and examined.

PROGNOSIS.—The prognosis of chronic tuberculosis of the lungs when the symptoms are at all advanced is very unfavorable, but the post-mortem examinations of so many individuals who have died of non-tubercular diseases show the presence of old tubercular lesions which have apparently ceased to be of grave import, that we must acknowledge that it is possible for many cases to survive the invasion of the disease.

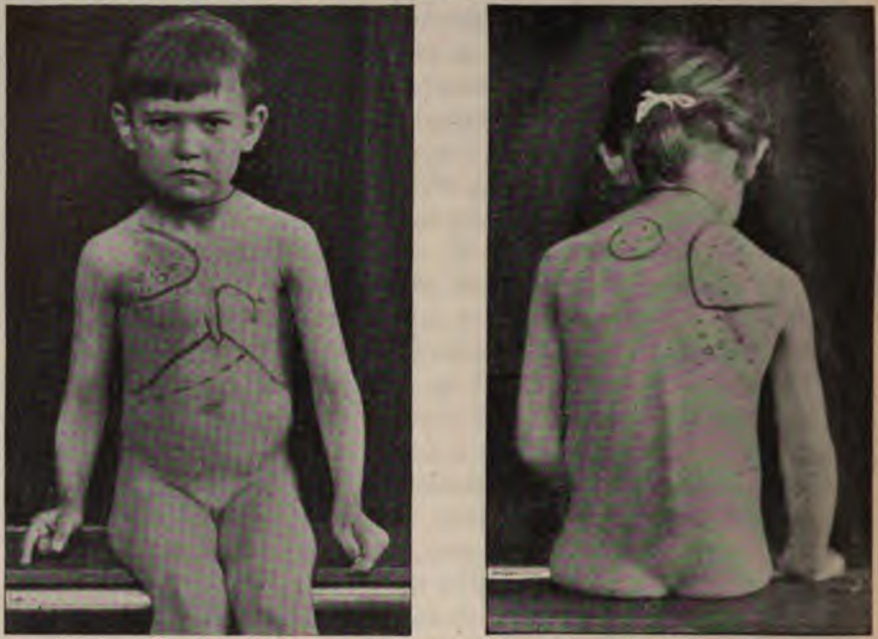
TREATMENT.—The treatment of chronic tuberculosis of the lungs is essentially climatic, and the children should be removed at once, if possible, from a climate where the altitude is low and the atmosphere damp and subject to great variations. Too high altitudes are also to be avoided. When the child cannot be removed to a more favorable locality, strict attention to its general hygiene and to its food will in some cases be followed by an apparent arrest of the tubercular process.

The treatment of chronic pulmonary tuberculosis especially calls for fresh air and sunshine. The food should be given at regular intervals five or six times in the twenty-four hours, and should be adapted to the digestion of the especial case. There are no drugs which are of much value in this disease. Cod-liver oil is commonly given, and in many cases seems to be tolerated by the stomach, and to be even agreeable to the child. In my experience, however, pasteurized twenty-four per cent cream with five per cent. lime-water is equally efficacious, and to most children much less distasteful. When the appetite is poor a nerve tonic, such as tincture of nux vomica, is often found to be beneficial, and when there is considerable anæmia the tartrate of iron and potash is indicated.

The following case (Fig. 100, p. 398) illustrates chronic tuberculosis of the lung following an acute infectious disease:

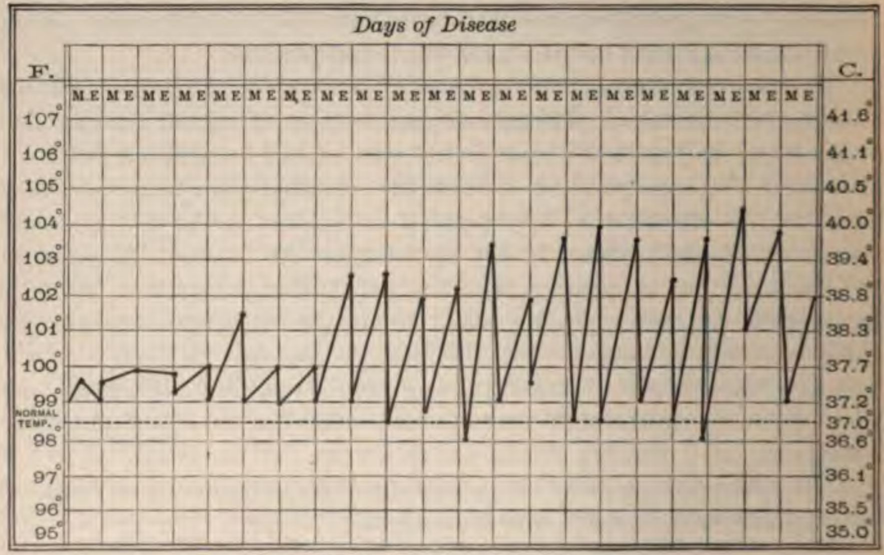
A girl, eight years old, had a history of tuberculosis in her family. She had an attack of pertussis when she was six years old, and some months later an attack of measles. Following the attack of measles she began to have headache, cough, and expectoration. She complained of pain in her chest and abdomen, and of chilly sensations, and progressively lost in weight and strength. A physical examination showed the skin to be dry and harsh and the heart normal. The left lung in front appeared to be normal. Behind over a small area at the upper part of the lung there were dulness, broncho-vesicular breathing, and some fine moist râles. Over the right upper lobe in front and behind there was dulness, and the expiration was prolonged

Fig. 100.



Chronic tuberculosis of the lung. Female, 8 years old.

CHART 5.



Chronic tuberculosis of the lung.

and high-pitched. Over the dull region were heard medium and fine moist râles. The borders of the areas of dulness are indicated by black lines, and the râles by black spots. The area of cardiac dulness, the lower part of the sternum, and the lower border of the ribs are indicated by dark lines, and the edge of the liver, which seemed to be somewhat enlarged, by an interrupted line.

The chart shows the irregular temperature which is commonly seen in cases of chronic tuberculosis of the lungs, and is of the remittent type.

The child was gradually failing and the signs of disorganization of the lung were slowly progressing.

TUBERCULOSIS OF THE PLEURA.

Tuberculosis of the pleura is a common affection in general tuberculosis and in tuberculosis of the lungs. The symptoms and treatment do not differ from non-tubercular pleuritis, but hemorrhagic exudations are rare in early childhood.

TUBERCULOSIS OF THE GASTRO-ENTERIC TRACT.—Tuberculosis of the gastro-enteric tract occurs most often in connection with and following tuberculosis of the lungs, the infection being due to the swallowing of sputum containing the tubercle bacillus. In rare cases the bacilli may find their entrance in milk or through food. Tuberculosis of the œsophagus, stomach, and duodenum is exceedingly rare. The œsophagus probably escapes infection owing to the fact that the sputum containing the bacilli remains for so short a time in contact with its walls, which are also protected by their many layers of epithelium. The stomach probably owes its protection to the presence of the gastric juice.

TUBERCULAR ILEO-COLITIS.

ETIOLOGY.—Tuberculosis of the intestine is not so common in infancy as in older children. In the latter, however, it is met with quite commonly, especially in the middle period of childhood. The disease may be primary in the intestine, but this is very rare. At the Boston Children's Hospital I have had one case in which the tubercular lesions were confined to the intestine and to the mesenteric nodes. In this case Professor Councilman considered that the evidence was in favor of the intestinal tubercle antedating the tubercle of the nodes. In the great majority of cases tubercular ileo-colitis is secondary to tuberculosis elsewhere, and in such cases is followed by tuberculosis of the mesenteric glands. The most common source of infection, however, in all parts of the gastro-enteric tract, is from the lung, by means of the sputum which has been swallowed.

PATHOLOGY.—The tubercular lesions are most common at the lower end of the ileum, next in the rectum, and then in the colon. In tuberculosis of the intestine the corresponding lymph-nodes are always affected. The tubercular lesions almost invariably occur in the form of ulcerations. These ulcers begin both in the solitary follicles and in Peyer's patches. The ulcers in the solitary follicles are small. In Peyer's patches they may be large, involving the entire extent of the patch. They also show a tendency to grow in the direction of the lymphatics, so that an ulcer may extend around the entire circumference of the intestine. The tubercular ulcer has the following characteristics: in contradistinction to the typhoidal ulcer, the long diameter of which coincides with the long axis of the intestine, the tubercular ulcer is transverse to the long axis, rarely ovoid, and often irregular in outline. The edges overhang and the base is infiltrated, and in many instances small tubercles may be visible to the unaided eye on the peritoneal surface at the base of the ulcer.

Fig. 101 represents a specimen of tubercular disease of the intestine which occurred in the practice of Dr. Northrup.

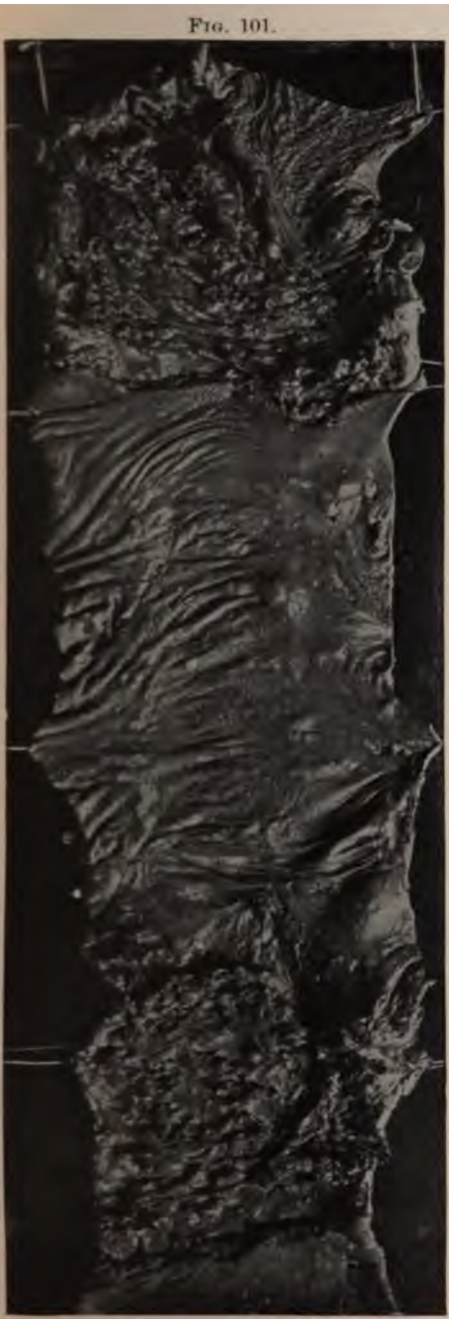
A girl eight and a half years old was attacked two months before her death with chills, fever, and prostration. The temperature at first varied from 38.8° to 39.4° C. (102° to 103° F.), but as the disease progressed the temperature gradually fell. There was rapid emaciation, the abdomen was sunken at first, but later became tense. There were pain, tenderness, and resistance in the right inguinal region. The submaxillary, cervical, and inguinal lymph-nodes were enlarged. The urine contained albumin and hyaline casts. There was diarrhœa. The autopsy showed the lungs to be normal. The bronchial and retro-peritoneal lymph-glands were enlarged and cheesy. The colon showed two large sloughing ulcers, one in the region of the cæcum and the other in the ascending portion. They were transverse to the axis of the colon, and their edges were overhanging. The entire membrane was thickened, and there was some follicular ulceration.

Fig. 102, I. and II., represent portions of intestine taken from a girl two years and eight months old. She had had diarrhœa occasionally for a year and also convulsions. She died soon after entering the hospital. The autopsy showed extensive ulcerations in the small intestine (I.), and a large ulcer in the cæcum (II.). There were tubercular ulcers in the middle third of the colon. The peritoneal surface showed miliary tubercles. The mesenteric and bronchial lymph-glands were markedly enlarged and cheesy.

Symptoms.—The symptoms of tubercular ileo-colitis are varied and indefinite. The most common symptom is a persistent diarrhœa. The diarrhœa, however, does not correspond to the extent of the lesions, as large ulcers may exist and constipation be present, especially if they are in the ileum.

Diagnosis.—In cases of primary tuberculosis of the intestine, the only certain means of determining the tubercular character of the disease is to find the tubercle bacillus in the discharges. When the disease is secondary to tuberculosis elsewhere, the tubercular involvement of the intestine may be suspected, when at any time during the course of the disease the infant is attacked with diarrhœa of an obstinate nature. In these cases the disease can only be established by finding the tubercle bacillus in the discharges. It is exceedingly difficult to do this when the stools are watery. It has been suggested that in order to simplify the search for the specific organism, sufficient opium should be given to produce a constipated movement, so that the smears may be taken from the mucus which is scraped from the tubercular ulcers and clings to the hard fæcal masses as they pass downward along the course of the intestine.

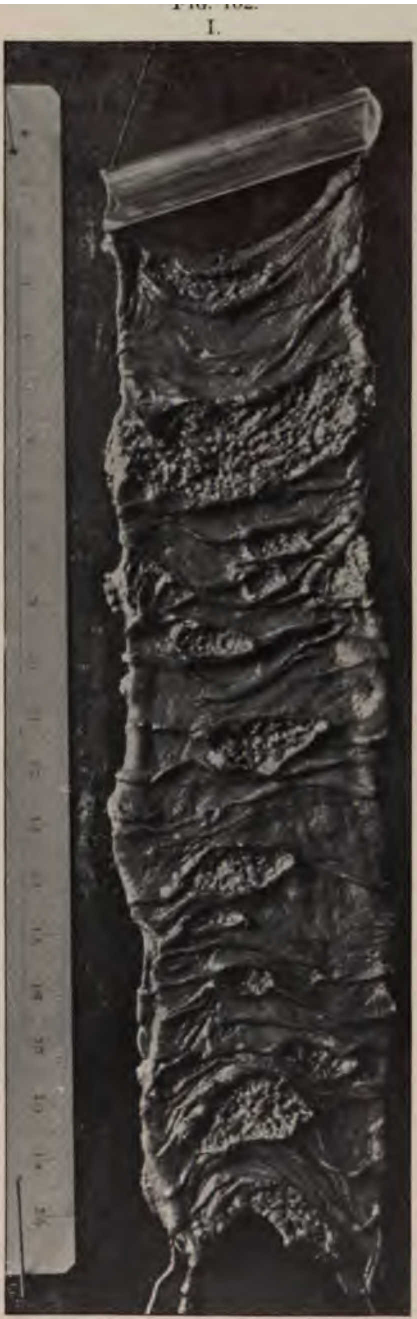
FIG. 101.



Tubercular ulcers of colon. Female, 8½ years old. Museum of the College of Physicians and Surgeons, New York.

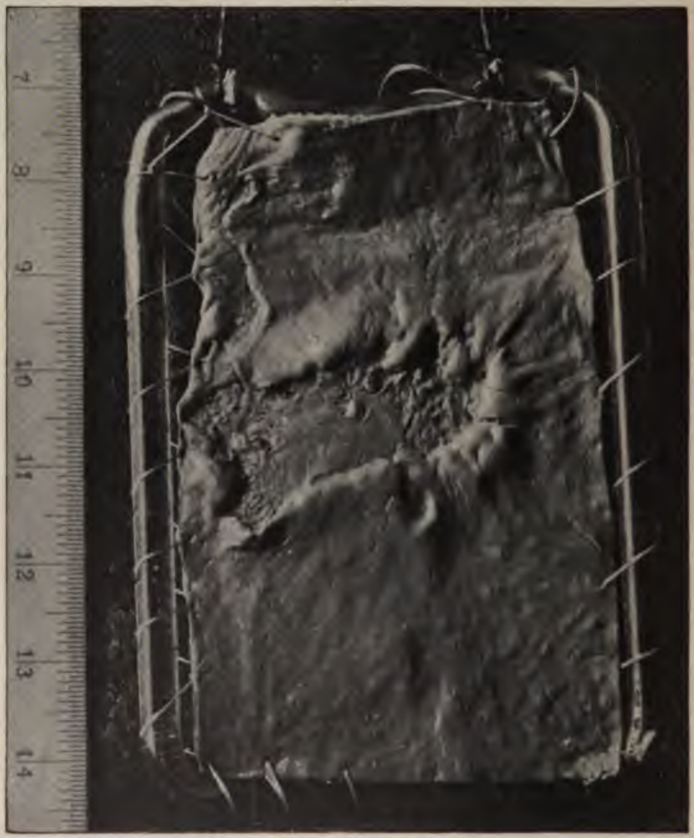
FIG. 102.

I.



Tubercular ulcers of small intestine. Female 2¾ years old. Museum of the College of Physicians and Surgeons, New York.

FIG. 102.
II.



Large tubercular ulcer of cæcum.

differential diagnosis is to be made from chronic non-tubercular
is. In the latter condition the history usually shows that the dis-
begun with an acute attack, while in tuberculosis the onset has
idious. Markedly enlarged mesenteric nodes and the evidence
ulosis elsewhere are important as indicating a tubercular cause
testinal symptoms.

NOSIS.—The prognosis is very unfavorable, and death may occur
om the severity of the intestinal symptoms, or, more rarely, by
on or hemorrhage. The prognosis, however, largely depends on
he tubercular disease to which the intestinal tuberculosis is sec-

TMENT.—The treatment is the same as that described for non-
r ileo-colitis on page 823.

TUBERCULOSIS OF THE PERITONEUM.

rculosis of the peritoneum may occur in both infants and chil-
a marked inflammatory condition, either with or without ascites,
ommon in children than in infants. In infants it is more com-
ave a miliary tuberculosis of the peritoneum in the course of a
uberculosis which does not, as a rule, show abdominal symptoms.
en the peritoneal inflammation may be so markedly localized as
tute a disease of itself, *tubercular peritonitis*.

OGY.—The original source of the tubercular process is often ob-
It may be a primary infection of the peritoneum, but most com-
secondary to tuberculosis of the mesenteric nodes. It may also
dary to tuberculosis of the intestine, lungs, lymph-nodes in vari-
of the body, and other organs. It may arise in the course of
us infectious diseases, especially the exanthemata. Tubercular
is may be *acute* or *chronic*, but the most common form met with
en, and the one which usually represents the disease clinically is
nic.

OLOGY.—The process consists in the formation of miliary tuber-
he peritoneal surface, which give rise to opaque cheesy thicken-
n nodular, with firm adhesions of the adjacent surfaces. An
n into the peritoneal cavity is usually present, the quantity gen-
ing considerable and sero-purulent in character. The ascites
wever, be serous, or merely purulent.

disease may occur in three forms. (1) A miliary tuberculosis
tes acute or subacute in its course. (2) A fibrous form in which
nay be, but is not usually, present. This form is essentially
There is a formation of a fibrous tissue with matting of the
of the omentum and mesentery, and not much tendency to
or breaking down. (3) An ulcerative form, which is a later
he two forms just described, and in which occur large tubercular
with caseation and softening. The lungs in this form are affected.

It is not so chronic as the fibrous form (2), but it is characterized by more fever and more severe symptoms.

SYMPTOMS.—The symptoms of tubercular peritonitis vary according to the extent and character of the lesions. When they consist of a miliary tuberculosis of the peritoneum, with only a slight inflammatory condition, they are usually secondary to a general tuberculosis, and the abdominal symptoms are insignificant. When this form occurs in young infants the later manifestations of abdominal disease are seldom seen, as the infant usually succumbs to the infection before the symptoms have had time to develop. When the disease is acute, the symptoms, as a rule, develop in connection with, or most often following, the symptoms of tuberculosis elsewhere, especially in the lung, and do not differ materially from those of acute non-tubercular peritonitis. When the disease is of the subacute and chronic form the symptoms vary according to the presence or absence of ascites as a prominent condition. Here, again, the symptoms correspond to the character of the lesions. In either case a tubercular process is going on, but in one it results in a large effusion of fluid, while in the other the more advanced tubercular lesions develop with no fluid at all, or with only a small amount.

The initial symptoms of tubercular peritonitis, with gradual development of ascites, are usually ill defined. There is a gradual loss of appetite and flesh, with occasional abdominal pain, which, as a rule, is not severe in character. There is seldom any tenderness of the abdomen. Vomiting is not a marked symptom. Attacks of diarrhœa are common and are apt to be paroxysmal. There may be constipation. The temperature is at times raised, especially in the latter part of the day, but, as a rule, is moderate,—37.2° to 38.8° C. (99° to 102° F.),—and may be so nearly normal as to simulate ascites due to obstruction. After these general symptoms have lasted for a number of weeks, the abdomen is noticed to be distended. A physical examination may show that there is nothing abnormal in the thorax, and that the morbid condition is confined entirely to the abdomen. At first the abdomen is resonant on percussion, but later fluctuation is detected. While for a time there is resonance in the region of the umbilicus when the child is lying on its back, and a change in the area of resonance with a change of position, this area of resonance gradually grows less. The fluid, however, may be encapsulated by adhesions and thus the area of dulness and resonance be less distinctly followed. As the disease progresses and the fluid increases, the whole abdomen becomes much distended and tense, the abdominal veins prominent, and the dulness and fluctuation diminished. In this higher grade of ascites the umbilicus is at times found to be pushed out by the fluid.

Clinically separated from the class of cases with ascites as the prominent symptom, are the still more chronic cases in which ascites is not prominent, but in which there are tubercular processes which, because of certain mechanical conditions, result in a set of symptoms induced by pressure, such as œdema, digestive disturbance, and renal congestion. The general symptoms are about the same as in the ascitic form just described, the difference being in the conditions found on examining the abdomen. Possibly the onset is still more insidious than in the other form, and the temperature more likely to be normal. The abdomen may be distended and tympanitic, or, again, varying areas of dulness may be detected, depending usually on tuberculous masses or tuberculous thickening of the greater omentum. There may at times be a certain amount of fluid which can be detected by fluctuation and change of position. Although there may be abdominal pain and tenderness, yet usually these symptoms are absent, and sometimes markedly so, even when the process has gone on to a purulent ascites.

The cases in which the more advanced tubercular lesions, such as ulceration and breaking down of the tubercular growths, have taken place are usually secondary to advanced lesions elsewhere, as in the lung, and do not properly constitute a localized tubercular disease. In these cases, however, the temperature is raised, a hectic condition is commonly present, the wasting is extreme, and all the symptoms irregular, so that when these symptoms become marked in the other forms of tubercular peritonitis just described, the presence of a general tuberculosis should at once be thought of.

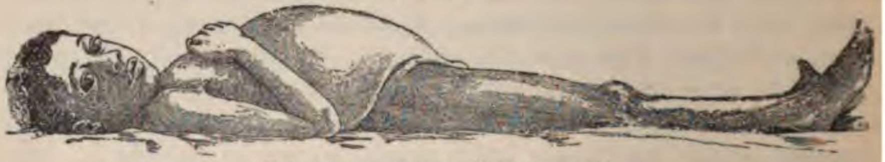
DIAGNOSIS.—In a case in which the symptoms which have just been mentioned are present the diagnosis is not difficult. Occasionally, however, there are no definite signs by which a diagnosis can be made, the only tangible sign being a seeming abdominal tumor, the resemblance of which to other abdominal tumors is so close that the diagnosis can only be made by laparotomy. We should remember, however, that most cases of doubtful abdominal tumors in children are tuberculous. In a case in which there is considerable ascites, it should be recognized that while the most frequent form of ascites in adults is obstruction of the portal circulation, as in cirrhosis, in young children, on the contrary, portal obstruction is rare, while ascites from tubercular peritonitis is exceedingly common, and is the disease which should at first be thought of. The greatest difficulty arises in differentiating simple non-tubercular peritonitis with ascites from the tubercular form. It is often impossible to do this without an exploratory paracentesis. If, however, there is evidence of tuberculosis elsewhere, or if the fluid is encapsulated, or if there is considerable fever, the process is most likely to be tubercular. In the cases of tubercular peritonitis where there are tubercular lymph-nodes without ascites, the diagnosis can only be made if irregular masses can be detected in the abdomen, accompanied by fever, wasting, and possibly some abdominal tenderness not accounted for by disease elsewhere. If tubercular disease is detected in the lungs, the diagnosis is still more assured. In many cases, however, the diagnosis can only be made by abdominal paracentesis. The diagnosis of the advanced cases, in which ulceration has taken place, is usually to be made as part of a tubercular condition of other organs, especially the lung.

Prognosis.—When tubercular lesions of other organs are present the prognosis is bad. In those cases in which the tuberculosis is apparently confined to the abdomen, the prognosis has been much improved since the employment of laparotomy for treatment. Any of these cases may, however, eventually die from a later infection, as of the brain or lung When untreated the prognosis of tubercular peritonitis is very variable In some cases the disease after a number of months retrogrades and th patient recovers. In most instances the child becomes more and mor wasted, the fever becomes more pronounced, the diarrhœa continues, th emaciation becomes extreme, and the child dies usually of exhaustion The surgical treatment of the disease has made the prognosis much mor favorable.

Treatment.—The general treatment of tubercular peritonitis does not differ from that of the non-tubercular form (see page 843). The essential treatment, however, in the cases which are supposed to be localized is surgical, especially when there is ascites of any amount. In some cases opening the abdomen and evacuating the fluid will not only give relief, but will produce a permanent cure. In my experience at the Boston Children's Hospital this procedure has been in a large number of cases followed by a complete arrest of the disease. Cases of marked tubercular peritonitis in which laparotomy had been performed, have been reported as perfectly well some years after the operation.

Fig. 103 represents a colored boy, nine years old, with tubercular peritonitis. His father died of phthisis. The boy had never been strong, but had suffered no acute illness. Three weeks previous to my seeing him, he began to have diarrhœa, and soon after, enlargement of the abdomen. There was no pain, vomiting, nor cough. He had lost greatly in weight and was much emaciated. His temperature was 38.3° C. (101° F.). The abdomen was much distended and gave a distinct wave of fluctuation. Physical examination showed nothing else abnormal.

Fig. 103.



Tubercular peritonitis. Male, 9 years old.

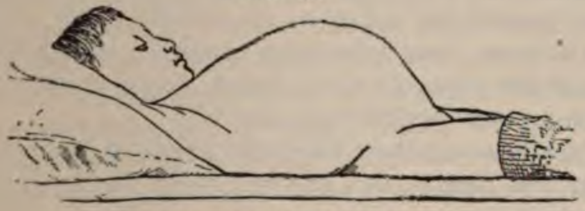
Laparotomy was performed and the fluid evacuated. Tubercle bacilli were found in the peritoneal tissue. When seen six months later the wound had healed perfectly, and he was strong and well.

Another case was that of a boy, two years old, and is especially interesting in regard to the diagnosis.

He had not had general symptoms of serious import, but had lost slightly in weight, appetite, and strength. From time to time for six months he complained of abdominal pain and tenderness. An examination of the abdomen showed a hardened, slightly irregular mass extending directly across the abdomen from one side to the other, 5 cm. (2 inches) above and the same distance below the umbilicus. It was not especially tender on pressure. Nothing else abnormal was detected about the child. The line of percussion did not change when he was lying on his back. There was no evidence of ascites. Laparotomy was performed and a mass of cheesy nodules matting together the intestine was found. An examination of a portion of this mass showed the presence of the tubercle bacillus. No fluid was present. The child recovered.

The next case was that of a boy, four years old, brought to the hospital with the extreme distention of the abdomen which is represented in Fig. 104. A physical examination showed nothing abnormal except in the abdomen, which was dull on percussion and showed fluctuation in every part. The child had gradually lost in weight, appetite, and strength. Laparotomy was performed and a large amount of ascitic fluid evacuated. Tubercle bacilli were present in the diseased peritoneum. The wound healed, but in the course of a few weeks the fluid re-accumulated, and laparotomy was again performed. Some weeks after the second operation no fluid could be detected. There was no recurrence of the ascites, and the child recovered completely.

FIG. 104.



Tubercular peritonitis. Male, 4 years old.

The next case is that of a boy, eleven years old. He had been perfectly well until four months before coming to the hospital, when he began to lose in weight and appetite and to show an increase in the size of his abdomen. Although he was not especially emaciated, he had lost in flesh and was pale. The circumference of the abdomen was 76.4 cm. (30 inches). On physical examination, nothing abnormal was detected in any of the other organs. For some months before the boy showed peritoneal symptoms, he had been drinking the milk of a tubercular cow. Laparotomy was performed and a large amount of serous fluid of a dark yellow color was removed. The peritoneum was found to be thickly studded with minute tubercles, and tubercle bacilli were demonstrated. The peritoneal cavity was irrigated and drained. He recovered completely from the operation, and when examined four years later, was found to be well and strong, and to show no signs of tubercular disease.

TUBERCULOSIS OF THE CEREBRO-SPINAL SYSTEM.—Tuberculosis of the cerebro-spinal system usually takes the form of an infection of the meninges (tubercular meningitis), and is found most commonly in the meninges at the base of the brain. As a rule, the disease is never so extensive in the spinal as in the cerebral meninges. Sometimes, though very rarely, a growth of miliary tubercles in the meninges extends into the tissues of the brain. The tubercle bacilli themselves are usually confined to the brain, but they may extend to the lungs and produce definite foci of embolic pneumonia.

TUBERCULOSIS OF THE BRAIN.

PATHOLOGY.—Tuberculosis of the brain may occur in the form of scattered miliary tubercles, or these tubercles may occur as nodular masses of various sizes formed by aggregations of miliary tubercles. These nodular masses may be single, but are more commonly multiple, and are found in both the cerebrum and cerebellum, but most frequently in the latter. They are occasionally found in the crus.

SYMPTOMS.—Tubercular lesions of the brain are always terminal. The scattered miliary tubercles are usually a part of a general miliary tuberculosis, and usually do not produce any special cerebral symptoms. When, however, masses of tubercles large enough to be considered tumors are present, they present the symptoms of cerebral tumor as described on page 978. In some cases tubercular tumors of considerable size have been found at the autopsy, when during life no symptoms have been noted. Such a case is described on page 980.

PROGNOSIS.—Although these tubercular lesions of the brain may remain latent during long periods, as a rule they result fatally, so that the prognosis is very bad.

TREATMENT.—The use of drugs in these cases is not of very much benefit. Surgical interference has proved of value when we are assured that a tubercular condition is present.

TUBERCULAR MENINGITIS.

ETIOLOGY.—Tubercular meningitis is a tubercular infection resulting in an inflammation of the pia mater. It is not a primary disease of the meninges, but is always a terminal lesion produced by the tubercle bacillus, which, originating elsewhere, is carried to the brain by the lymphatics or the blood-vessels. In infants the tubercular meningitis is usually secondary to a general tuberculosis or to tubercular lesions in the lungs. In older children it is apt to be secondary to tuberculosis of the lymph-nodes, bones, or joints. The tubercular form of otitis is not uncommon, and may be the starting-point of a meningeal infection. Tubercular meningitis is the most common form of meningeal disease in children. It occurs most commonly in early life, runs a subacute course, and is invariably fatal. It occurs more commonly between the ages of two and seven, than at any other period of life. It is rare in the first year of life, especially in the early months; the number of cases increases rapidly in the second year and decreases as rapidly after the eighth year. It is comparatively so rare in adult life that out of a large number of adult patients that I met in my service at the City Hospital only a few cases of tubercular meningitis came under my care during a service of ten years. In a large number of cases there is a tubercular history of one or both parents.

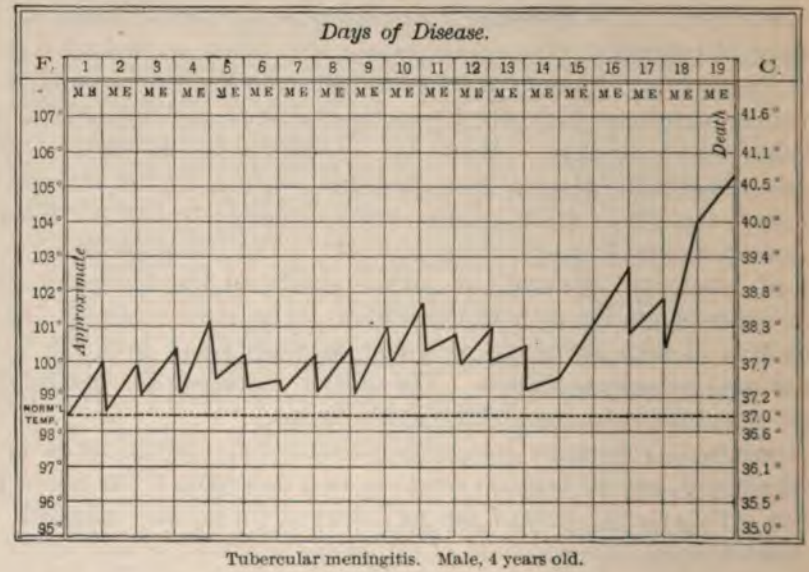
PATHOLOGY.—A knowledge of the general pathology of tubercular meningitis is of great practical importance in acquiring a clear picture of the disease. Although the nidus of the tubercle bacillus which produces the pathological lesions of tubercular meningitis is in some other part of the body, and the lesions of the brain and its meninges are always secondary, yet, as the clinical characteristics of the disease are those of a primary cerebral nature, only the morbid lesions which occur in the brain will be described.

The macroscopic pathological condition which is seen in the brain as a result of the action of the tubercle bacillus is a growth of miliary tubercle in the meninges. This growth is especially marked in the meshes of the pia mater along the course of the blood-vessels at the base of the brain. These small granulations are conspicuously numerous in the choroid plexus, and cause great irritation in the neighboring parts. The irritation is followed by an exudation of greater or less extent into the ventricles. Accompanying this there is also a fibrino-purulent exudation between the pia mater and the cerebral convolutions at the base of the brain, notably in the fissures of Sylvius, but at times covering the whole convexity. This inflammatory exudation is usually confined to the meninges, but it may extend into the tissue of both brain and cord and over the upper surface of the cerebellum. A marked characteristic of the disease, and one which tends to distinguish it from other forms of meningitis, is the extension of the exudation from the meninges around the sheaths of the cranial nerves. The nerves apt to be affected are the auditory, optic, and the fifth nerve. The amount of exudation is not proportionate to the number of tubercles. The ventricles are sometimes so distended as to burst the septum. Pressure is thus brought upon the central portions of the brain, involving especially the optic thalamus, the corpus striatum, and the corpus callosum. While the symptoms vary in different individuals and at different ages, the pathological lesions, on the other hand, with the exception of their location, are comparatively stable. What is of especial interest to us clinically, however, is that, although in a typical case of tubercular meningitis in middle childhood the symptoms, as a rule, correspond to the pathological lesions, yet in some cases we find an entire lack of such symptoms as would naturally result from the widespread and prominent lesions. The spinal meninges are occasionally involved in tubercular spondylitis (Pott's disease).

SYMPTOMS.—Tubercular meningitis presents many irregularities in its manifestations, and its typical symptoms vary according to the age of the patient. By careful study of the pathology of tubercular meningitis we can almost deduce the sequence of symptoms which we should expect to meet with in the middle period of childhood, a period when the disease is seen in its most typical form. In fact, in the great majority of cases occurring between the ages of two and eight years this sequence is very striking. As we are dealing with a symptom of general tuberculosis, we should expect to find in the early stages of the disease that the nutrition is affected, that there are a lessened appetite, loss in weight, anæmia, and, in fact, symptoms which warn us that something is affecting the child's general health. This condition may last for many weeks, or even months, varying as to the time when the tubercle bacillus has left its original nidus and migrated to the cerebral meninges. Only after this has occurred do we begin to get symptoms of cerebral irritation. The child then becomes peevish and capricious, and is in some cases easily frightened. As the tubercular growth increases and causes further congestion of the blood-vessels, the sleep is disturbed; the child complains of dizziness and slight evanescent pains in the head; it staggers slightly in its walk (static ataxia); sometimes it cries out sharply, especially at night (hydrocephalic cry). Vomiting, apparently not connected with the food, and usuall without nausea, is a common symptom. It may occur only once o several times, or it may last for a number of days. These are symptom of irritation of the nervous centres, and may last for a week or two, ac cording to the development of the pathological lesions. Progressiv emaciation becomes prominent, and the increasing apathy is very notice able.

The temperature is usually moderately raised, 37.2°–37.7°–38.3° C (99°–100°–101° F.), but on some days it rises a degree or so higher, an just before death a considerable elevation may occur. Chart 6 shows th

CHART 6.

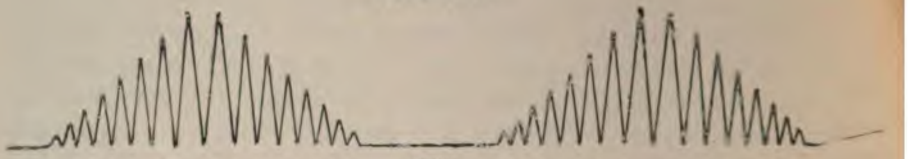


Tubercular meningitis. Male, 4 years old.

temperature of a child four years old. It represents very well what will usually be met with in uncomplicated cases of tubercular meningitis. It is impossible to determine the exact day of the beginning of the disease in such an affection as tubercular meningitis, so that the first day marked on the chart is merely approximate and serves as a starting-point to show the character of the temperature. The pulse at first is somewhat accelerated, but soon becomes slower than normal, and is apt to intermit. In determining whether the pulse is slow, the age of the child must be taken into consideration. (See page 71.) The respirations may in the early part of the disease be quickened, and at times are of a sighing character. Obstinate constipation is a common symptom. The urine is scanty. Hyperæsthesia of the skin, with occasional waves of congestion, especially of the cheeks, is sometimes met with. The pain in the head increases, and the child is apt to hold its hand to its head. Drowsiness, at first slight, soon becomes very marked, and gradually changes to stupor, difficulty in arousing the child becoming greater and greater. The child is apathetic and lies in bed, refusing to eat. There is photophobia, and the pupils may, early in the disease, be contracted. Tubercles in the fundus of the eye are rarely seen during life. The eyes have a fixed, expressionless look. Abdominal pains are quite frequent, and depression of the abdomen (boat-shaped) is noticed in a certain number of cases. Drawing the finger over the skin usually produces a bright red line, which becomes in a few minutes quite intense, and lasts perhaps ten or fifteen minutes, which is much longer than would be the case in a healthy child. This phenomenon is called the *tache cérébrale*, and is quite frequently met with in tubercular meningitis, although it may be absent. This sign is, however, in no sense typical, and is seen in a number of other diseases. Kernig's sign may or may not be present. The child at this stage of the disease is apt to roll its head on the pillow almost continuously. If diarrhœa appears, we should suspect tuberculosis of the intestine.

When the pathological irritation has gone on to exudation, we begin to get symptoms of pressure. Sopor comes on rapidly, and the child can no longer be aroused. Strabismus, nystagmus, and ptosis may appear. The pupils are dilated and irregular, respond slowly to light and later not at all. The cornea becomes clouded. The Meibomian secretion is sometimes markedly increased. Muscular twitching may occur. Convulsions, generally partial, and of a rather mild type, set in. Rigidity of the neck and, at times, opisthotonos develop. Movement of the head and trunk and pressure over the spinous processes may cause pain. At times paralysis of the arm, or of the arm and leg (hemiplegia), and interference with sight (optic neuritis), may be noticed. The cutaneous reflexes are diminished and the deep reflexes of the arms and legs are, as a rule, increased. The paralysis which in some cases occurs early in the disease is not necessarily permanent. At this stage the pulse becomes markedly slow and irregular, 50–60–70, and it is very common to find an intermission in the pulse, although this must not be considered as diagnostic of tubercular meningitis. The respirations are not perceptibly diminished at first, but soon become slow, 10 to 15 in a minute. A peculiar form of respiration, called Cheyne-Stokes, is usually seen at this stage of the disease. The type of respiration is characterized by complete or almost complete cessation of the respiratory movements for a number of seconds. This is followed by a faint return of the respiratory movements, which gradually increase in depth, rising for five or six inspirations and then fading away again so as to be imperceptible. Chart 7 represents this type of respirations occurring in the third week of the illness of a child four years old who died of tubercular meningitis.

CHART 7.



Cheyne-Stokes respiration. Tubercular meningitis. Child, 4 years old.

A heightened temperature in tubercular meningitis indicates a complication of some kind, such as pulmonary tuberculosis, pneumonia, or tuberculosis of the intestine. At the end of the disease, however, the temperature rises rapidly, as do the pulse and respirations. Hearing, taste, and smell seem to be unimpaired for some time. The position which children with tubercular meningitis often take is somewhat characteristic. In all forms of meningitis they are apt to bury their heads in the bedclothes, and there is often spasmodic retraction of the head. They are inclined to lie with their knees drawn up, and there may be spasmodic opisthotonos. A striking feature which not infrequently occurs in the course of these general symptoms is a partial return to consciousness after the child has been lying in a stupor for several days. This phenomenon often induces the parents, and sometimes even the physician, to entertain hopes of improvement. It is, however, always delusive, for it has no favorable significance, and is soon followed by a more profound state of unconsciousness. These symptoms which been have mentioned do not, of course, always appear together, but may be present in different groups, varying with the individual. All the symptoms may disappear temporarily.

The duration of the disease varies. It is usually from three to six weeks, but may last only one week, and again eight or even twelve weeks. The cases of shorter duration usually present more active and severe symptoms, and ones more difficult to differentiate from simple acute meningitis. Death may be preceded by continued convulsions for several hours, but usually the pulse becomes weaker, the temperature rises, the child can no longer be made to swallow, and, according to Sachs, dies from paralysis of the cardiac and respiratory centres.

Infantile Tubercular Meningitis.—SYMPTOMS.—According to some extended observations made at the hospital in Stockholm, infantile tubercular meningitis is characterized in the first year by an absence of prodromata, the sudden development of acute symptoms, a short course, and a fatal issue. The temperature is high,—38.8°–39.4°–40° C. (102°–103°–

104° F.) The respirations are quickened and comparatively regular,—30–40–50. The pulse is high,—130–140–150. Clonic spasms and strabismus often occur. Paralysis is quite frequent, and diarrhœa is present rather than constipation. Bulging of the fontanelles is usual. Sinking of the abdomen is rare. Vomiting may occur, but is not especially common. Sharp cries are occasionally met with. The differential diagnosis from non-tubercular meningitis is difficult. Sopor and coma at the end are frequent in both diseases. The duration is seldom more than a week. It may be only two days, yet in rare cases the infant, like the child, may live for a month.

During the second year the symptoms of tubercular meningitis become of an irregular type, sometimes assuming the character of those which are seen in the first year, but soon corresponding more nearly to those which are met with in the middle period of childhood.

DIAGNOSIS.—The diagnosis of tubercular meningitis in the middle period of childhood, and with the sequence of symptoms just described, is not difficult, but in the early days, or even in the first week of the disease, must necessarily be very obscure. When the process occurs in young infants the difficulty is still greater. It is by watching the course of the disease and the general grouping of symptoms, rather than by the consideration of any one symptom, or even one group of symptoms, that we are justified in making a definite diagnosis. The diagnosis, then, must, as a rule, be held in abeyance for many days. Reflex vomiting with a moderate temperature, irregularity and intermission of the pulse, apathy, and many other symptoms of tubercular meningitis I have seen in cases in which there were no serious cerebral lesions. The active development and sensitive condition of the nervous system in children are so exaggerated in comparison with those of adults, that whatever condition may be present, it is liable to produce so profound an impression on the child's nervous centres that actual disease of these centres is readily simulated. Thus, for days symptoms apparently cerebral may mask by their undue prominence the presence of the real disease.

As a rule, in most cases the distinctive picture of a beginning tubercular meningitis after the first year of life closely simulates that of a simple digestive attack. Reflex vomiting, continued for some time without adequate explanation, and progressive loss in weight and strength, with change in temperament, should awaken suspicion of the more serious disease. Marked constipation, apathy, and drowsiness, slowly increasing stupor, and slow, irregular pulse and respiration, with a moderate temperature, will strengthen the diagnosis. Confirmatory evidence of the presence of tubercular meningitis can be obtained in many cases by means of lumbar puncture. The differential diagnosis from other diseases and the presence of some form of meningitis can readily be made by the cerebro-spinal fluid, which contains some form of pathogenic organism and is usually cloudy. The cerebro-spinal fluid in tubercular meningitis contains cells which are essentially lymphoid in character, the polymorphonuclear leucocytes being much less numerous. In addition to this, the fluid is less turbid than in the other forms of meningitis. Cover-glass preparations may show the tubercle bacillus, and inoculation of the fluid will often produce tuberculosis in guinea-pigs.

In the other forms of meningitis, including the epidemic cerebro-spinal form, the cells are mostly of the polymorphonuclear variety, while the lymphoid cells are less numerous. The fluid is also more cloudy than in the tubercular form. In addition to this, some special organism may be found, such as the pneumococcus in cases of pneumonia, and the diplococcus intracellularis in the epidemic form.

Differential Diagnosis.—The differential diagnosis must be made between meningitis in general and other diseases, such as (1) diseases of the stomach, (2) anterior poliomyelitis, (3) pneumonia, (4) malaria, (5) typhoid fever, (6) syphilis, (7) nephritis, (8) epidemic cerebro-spinal meningitis, and (9) acute meningitis.

(1) *From Diseases of the Stomach.*—Unless the child is very young, symptoms due to acute gastric disturbance are, as a rule, not difficult to recognize after the first few days. We may at times, however, be suspicious of cerebral disease when in an infant there is continual vomiting and an elevated temperature, in whom there is no discoverable source of reflex irritation to account for the symptoms. This is especially the case if there is some irregularity of respiration and the pulse is slow. These may prove to be cases of tubercular meningitis in the first year of life. Again, however, they may be simply cases of reflex vomiting. As an illustration of this class of reflex gastric disturbance the following case may be cited:

A male infant, eight months old, was attacked with vomiting which lasted with short intervals for two days. There was apathy, and the pulse was slow and intermittent. The temperature was 37.2° C. (99° F.). There were irregular respiration and rapid emaciation. The patient made a perfect recovery in four or five days, and the case proved to be one of cyclic vomiting. The slow, intermittent pulse, and the moderate temperature, which would have been so alarming in an older child, led me in this case, as in others in the first year of life, to eliminate tubercular meningitis. In my experience this interpretation of symptoms has proved to be correct.

(2) *From Anterior Poliomyelitis.*—The following case of anterior poliomyelitis resembled tubercular meningitis:

A boy, eighteen months old, showed for over a week symptoms closely simulating those of tubercular meningitis. Obstinate constipation and apathy were present, followed by unconsciousness; there were also a marked *tache cérébrale*, distended fontanelle, irregular pulse, contracted pupils, eyes turned upward, and convulsive attacks. Finally, paralysis of the arms appeared, the general symptoms passed off, and the diagnosis of anterior poliomyelitis was readily made.

(3) *From Pneumonia.*—A certain number of both primary and secondary cases of meningitis have been proved to have been caused

by the pneumococcus lanceolatus, but the number as yet has been too few to formulate a symptomatology separate from that of the other forms of meningitis. The cases of pneumonia reported on page 682 warn us that we should hold our diagnosis in abeyance sometimes even for a week, and that the nervous symptoms arising in the course of a pneumonia may closely simulate those arising from meningitis.

(4) *From Malaria.*—Although we must admit that malaria closely simulates almost any disease, it is not usual to mistake the malaria of older children for tubercular meningitis. An examination of the blood for the presence of the plasmodium will generally make the diagnosis clear. In the first two years of life, however, malaria may affect so insidiously the general nutrition before its characteristic symptoms appear that some doubt as to the differential diagnosis may arise, as illustrated in the following case:

A male infant, twenty months old, with a history of tuberculosis on the mother's side, began to show symptoms of anæmia and malnutrition with no perceptible cause, such as improper food or bad general hygiene, to account for it. After two or three weeks it had attacks of unconsciousness lasting for hours; at other times drowsiness, with irregular pulse and respirations, was noticed. The temperature was 39.5° to 40° C. (103° to 104° F.). There were slight convulsions, and the fontanelles were distended. At first there was no periodicity of the symptoms, but a week later the attacks were evidently more pronounced every other day, while on the intervening days the infant was brighter. It lived in a malarial district. On the administration of quinine and on removing the infant to a non-malarial region, these symptoms entirely disappeared. The detection of the plasmodium would, of course, have determined the diagnosis in this case, but it could not be obtained.

Another case, which I saw in consultation, is also very instructive in warning us to be careful in making a diagnosis of tubercular meningitis in cases in which there is a possibility of malaria being the cause of the symptoms.

A male infant, fourteen months old, had always been well until fourteen days previous to the time when I first saw it. It then began to be fretful and to have diarrhœa. This condition continued for about a week, when it fell into a stupor, became very anæmic, and it was necessary to feed it by means of a dropper. At times it would cry out sharply. The temperature varied from 37.2° to 38.7° C. (99° to 102° F.). The respirations were usually regular, but at times were of the Cheyne-Stokes type. The pulse was about 120, sometimes regular, but at times intermitting. The pupils were sometimes contracted, but showed no irregularity. No other abnormal conditions were detected, but the abdomen was depressed. The *tache cérébrale* was very distinct.

On close inquiry I found that there was a slight periodicity in the symptoms, shown by a rise of temperature on each afternoon and followed by the stupor becoming somewhat less. Although the infant had been unconscious for a week, and was becoming weaker and taking less nourishment every day, yet, on the supposition that it might possibly be an obscure case of malaria, I decided that quinine should be administered in suppositories. On the next day a slight improvement was noticed during the afternoon. The infant appeared less comatose, but its temperature and pulse remained as on the previous days. On the following day, which was the second from the time that it had begun to receive the quinine, it rapidly became conscious and began to drink milk. On the following day it was reported to have had a restless night and two slight convulsions. Its temperature in the morning was 38.2° C. (100.9° F.), and the pulse was 115 and not intermittent. On the next day there was marked improvement in every way, and this continued without interruption for the following four days. The infant then continued to improve rapidly, the temperature and pulse becoming normal and later it was reported to be perfectly well.

(5) *From Typhoid Fever.*—In my experience in cases in which posit results are not obtained from the Widal reaction and from lumbar pu ture, typhoid fever in young children is the disease which, next to t non-tubercular forms of meningitis, is most likely to simulate and be m taken for tubercular meningitis. The differential diagnosis is given page 453. We may also have considerable difficulty in differentiating t bercular meningitis from the non-tubercular meningitis which may occ in the course of typhoid fever; this form of meningitis, though exceeding rare, may occur, as has been proved by finding the typhoid bacillus in th cerebro-spinal fluid by lumbar puncture. The extreme cerebral congestion which at times arises as a symptom of typhoid fever may also add fresh difficulties to the differential diagnosis.

(6) *From Syphilis.*—The history and general symptoms of syphilis are to be sought for where a syphilitic meningitis is suspected. The temperature is not especially high, and the symptoms are seldom acute. The pathology is said to be usually that of a chronic basic meningitis.

(7) *From Nephritis.*—In addition to the other diseases which may simulate tubercular meningitis should be mentioned nephritis, in which the symptoms of uræmia simulate, to a certain extent, those of tubercular meningitis. The urine should always be examined in doubtful cases of this kind. When uræmic symptoms resulting from nephritis are present the disease will be shown by such examination, and we are thus able to differentiate it from tubercular meningitis.

(8) *From Epidemic Cerebro-Spinal Meningitis.*—It is often quite difficult to differentiate the early stages of tubercular meningitis from those of cerebro-spinal meningitis. In typical cases, however, the diagnosis is not difficult, as the long prodromal period of tubercular meningitis, as a rule, does not occur in cerebro-spinal meningitis. The temperature in the latter disease is high in comparison with the former, in which it is moderate. In fact, all the symptoms of cerebro-spinal meningitis are markedly acute in comparison with those of tubercular meningitis, which is essentially a disease of a subacute character. The sudden onset, extreme hyperæsthesia and sensitiveness to sound, intense headache, and marked tenderness on pressure over the spine, so common in cerebro-spinal meningitis, are seldom met with in the tubercular form. The absence of leucocytosis points strongly to tubercular meningitis; if leucocytosis is present, it is of little value in the differential diagnosis.

(9) *From Acute Meningitis.*—The symptoms of meningitis in general

can, in the great majority of cases, be differentiated from other diseases, provided that we do not attempt to make the diagnosis too early.

Having determined that the disease is of cerebral origin, we must next differentiate between the tubercular and the other forms of meningitis by means of the broad rules of which I have just spoken, and which I have condensed and simplified by means of the following table:

TABLE 61.

CEREBRAL MENINGITIS.

Acute Form.	*Tubercular Form.*
Usually secondary (possibly primary).	Secondary.
Not hereditary.	Hereditary.
Acute.	Subacute.
Prodromata short, if any.	Prodromata long; decided.
Headache severe at once, with delirium early, and soon followed by somnolence.	Headache less severe at first, but gradually increasing; delirium less common and milder.
Photophobia extreme.	Photophobia not so marked.
Convulsions violent.	Convulsions less violent.
Temperature high.	Temperature moderate.
Pulse and respiration rapid.	Pulse and respiration slow and irregular.
Duration short.	Duration long.
Cerebro-spinal fluid turbid, with preponderance of polymorphonuclear cells.	Cerebro-spinal fluid less turbid, with preponderance of lymphoid cells.
Leucocytosis present.	Leucocytosis sometimes present, often absent.
Presence of specific organism in the cerebro-spinal fluid.	Presence of tubercle bacillus in cerebro-spinal fluid.

Transudation into the ventricles may occur in either form. The younger the infant the nearer the two forms approach each other in the similarity of their symptoms.

PROGNOSIS.—When we are sure of our diagnosis, I believe that in our prognosis we should give no hope of recovery whatever, except that in extremely rare cases a temporary remission may take place. The reported cases of absolute recovery from tubercular meningitis cannot but be looked upon with scepticism. Indeed, the acute forms of meningitis simulate the tubercular so closely that without post-mortem verification recoveries can be supposed to be possible, but can hardly be accepted as proved.

TREATMENT.—The treatment of tubercular meningitis up to the time when the diagnosis is established should be purely symptomatic; later we should make the child comfortable by every means in our power. As no case of tubercular meningitis has ever been proved to be cured by iodide of potassium or any other drug, it is useless and unwise to encourage ourselves and the parents by false hopes of good results arising from the administration of any drug whatever. Up to the present time our knowledge of the disease justifies us only in using drugs as palliatives for the child's suffering.

The following cases illustrate the different phases of tubercular meningitis. The first case illustrates a type of disease such as may occur in the first year of life:

A male infant, ten months old, had always been well and strong. For a few days before he was seen he had been rather dull and feverish, but had shown no other abnormal symptoms. He was evidently cutting some teeth at that time. On the day saw him, except that he was somewhat fretful and put his hands to his mouth though his gums were disturbing him, he seemed very well, and careful physical examination revealed nothing abnormal in the ear, throat, chest, or abdomen. On the d following my visit the slight symptoms of indisposition which he had previously show disappeared, and he played with a toy whistle, blowing it himself, and seemed to very well. This condition lasted for two or three days, when he became stupid and unconscious, and about the tenth day from the time he first came under my observation died in convulsions.

This case should impress upon us the difficulty of making a diagnosis in the ear period of a tubercular meningitis, and how guarded we should be in giving a prognos in young infants, even when the character of the disturbance is very slight.

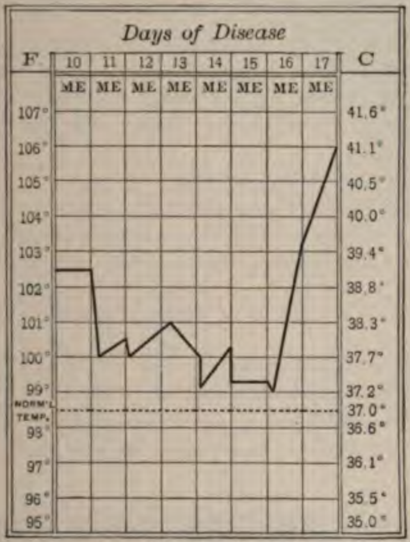
The next case illustrates a tubercular meningitis occurring in a child two and a half years of age:

The history of the case showed that the father's mother and the mother's mother and brother had died of consumption. When the patient was one year old he had measles; otherwise he had always been well. About two or three weeks before he was first seen it was noticed that the child slept more than usual. At that time he appeared to be feverish, his tongue was coated, but there was no nausea nor vomiting. A few days later he vomited once or twice during the day. The bowels were constipated. Eight days before entering the hospital he had a slight convulsion, and three days later he cried a great deal, as if in pain. Two days before entering the hospital he had a number of convulsions during the night, each lasting about ten minutes. On the following day the convulsions recurred. On the day he entered the hospital he began to have convulsions at three o'clock, which lasted about two and a half hours. At that time he was noticed to have marked internal strabismus of the left eye and slight strabismus of the right eye. The muscles of the neck were somewhat contracted. There was no paralysis of the extremities.

The pupils were equal, reacted to light, and were somewhat dilated. The conjunctivæ were injected, the left one especially so. Sensation was not impaired. The knee-jerks and ankle-clonus were absent. There was a marked *tache cérébrale*. The respirations were irregular and sometimes of the Cheyne-Stokes type. The child was unconscious and very pale. The heart's action was very rapid, sometimes as high as 200 beats in a minute. No souffles were detected. The temperature was 38.3° C. (101° F.). During the next day the child lay in a state of stupor. He continually moved the left forefinger and thumb, kept drawing the head to the left, and was very restless. He was reported to have cried all night and to have put his hand to his left ear. He lay with his eyes wide open, took nourishment well, and had less strabismus than when he entered the hospital. On the following day (about the thirteenth day of the disease) he became very restless, had sordes on the teeth, and his tongue was very dry. Examination of the ears showed nothing abnormal. The abdomen was somewhat retracted. The bowels were moved regularly, and the movements appeared to be well digested. He took about 90 c.c. (3 ounces) of milk every two hours. On the following day there was no especial change, except that the muscles of the neck were firmly contracted and the *tache cérébrale* came out more slowly than on the previous day. A slight paralysis of the left side of the face appeared on this day. The left eyelid moved rather slowly, and the left corner of the mouth seemed to drop a little. The pulse was irregular, of fair strength, and intermittent. He did not take his nourishment so well.

The comatose condition and other signs continued without improvement. The face became cyanotic, the respirations Cheyne-Stokes in character, the pulse irregular and ntermittent. The temperature gradually fell until it was 37.2° C. (99° F.) in the

CHART 8.



Tubercular meningitis. Male, 2½ years old.

morning of the sixteenth day of the disease, when it began to rise rapidly until it reached 41.1° C. (106° F.) in the evening of the seventeenth day, when the child died.

The autopsy showed the following lesions :

Heart.—The right ventricle was dilated and the valves were normal.

Lungs.—A number of small, flattened, gray masses were found in the pleura ; on section they were found to be miliary tubercles. Ther ight lung was adherent to the parietal pleura by strong fibrinous adhesions, beneath which were miliary tubercles, especially in the areas covering the ribs, the diaphragm, and the upper third of the sternum. A small number were also found in the substance of the lung. The bronchial glands were enlarged, one of them being 1.2 cm. (½ inch) in diameter. This gland on section was yellow and somewhat broken down.

Spleen.—The spleen was of about normal size and showed many flattened miliary tubercles. Beneath the capsule, on section, there were found numerous tubercles of varying size : the larger ones were yellow and the smaller ones gray.

Peritoneum.—There were found scattered all through the omentum, on the surface of the root of the mesentery, over the bladder, and particularly on the under surface of the right side of the diaphragm, numerous miliary tubercles. The lymph-glands of the mesentery were considerably enlarged, particularly beneath the stomach. On section they showed tubercles, most of which were quite large and had yellow, cheesy centres.

Intestine.—In the intestine about the ileo-cæcal valve there were several small ulcerations apparently in the process of repair. In the cæcum there were two narrow ulcers about 1.5 cm. (⅝ inch) long. The bases were injected. The walls were not broken down.

Liver.—Many rather large tubercles were found beneath the capsule of the liver. They were flat, but not cheesy.

Brain.—The convolutions of the brain were flattened. There was marked fibrino-serous exudation at the base of the brain, covering the optic commissures and the adjoining parts. The third nerve was chiefly injected. Many small tubercles were present in the fissures of Sylvius and over the convexities of the brain. In the right half of the cerebellum, just beneath the pia, about the centre of the base, was a yellow nodule about 6 mm. (¼ inch) in diameter. In the left lateral ventricle anterior to the velum interpositum was a similar nodule about 3 mm. (⅛ inch) in diameter projecting into the ventricles. Both ventricles were moderately dilated by the serous fluid. The ependyma was everywhere granular: this condition was due to small, gray, transparent tubercles. No tubercles were found in the third or fourth ventricles.

Kidneys.—The kidneys contained a few rather large grayish areas with here and there a yellowish speck.

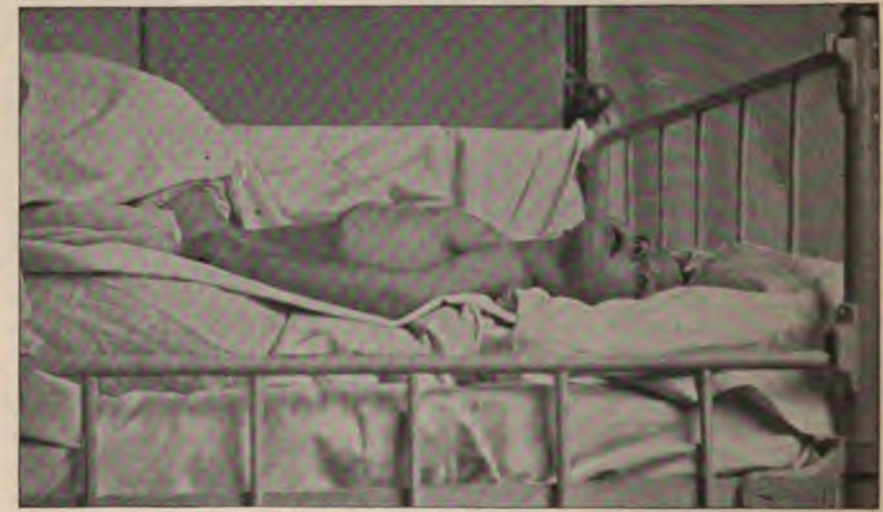
The *pathological diagnosis* of the case was—

Old tubercular ulcers of the intestine; chronic tuberculosis of the mesenteric and bronchial lymph-glands; solitary tubercle of the brain; miliary tuberculosis of the pia, lateral ventricles, pleura, lung, spleen, kidney, peritoneum, and liver.

Another case was that of a boy three years old.

There was no history of tubercular or syphilitic disease in the parents. Three weeks before entering the hospital, the child, who had previously been healthy, began to complain of pain in the abdomen, anorexia, and general malaise. Somewhat later it was noticed that the eyes would at times turn inwards and that the head would be drawn back. He was in this condition for two weeks before entering the hospital. On March 13 he was brought to the hospital, and was found to have a temperature of 38.4° C. (101.2° F.), a pulse of 120, not intermitting, and respirations 40. He was in a stupid and drowsy condition. His head was drawn back, and he would not lie on his back. The tongue was not coated. An examination of the heart, lungs, and urine showed nothing abnormal. An examination of the eyes showed the pupils to be dilated, but equal in size and reacting to light. There were internal strabismus of both eyes, an optic neuritis, and the beginning of an atrophy following the neuritis. The patellar reflexes were absent, and there was no ankle-clonus. The superficial reflexes were normal. There was no tenderness of the head or spine. An examination of the ear showed nothing abnormal. On March 16 he showed nystagmus with conjugate devia-

FIG. 105.



Tubercular meningitis. Male, 3 years old.

tion to the right or to the left, according to the side on which he lay. No *tache cérébrale* was present. On the 17th an erythematous congestion was noticed on the right cheek, and he became still more somnolent. On the 18th the head was much less retracted. He had vomited once during the night and once in the morning. On the 21st he had a convulsion, which was the first that had occurred during the course of the illness. He was also found to have partial opisthotonos. The legs did not participate in the contraction, but the head was drawn back almost to the buttocks. His respirations became Cheyne-Stokes in character. On the same day he had four or five convulsions, and remained in a condition of opisthotonos in the intervals between them. These convulsive attacks lasted about half a minute each, and the intervals between them were about four minutes. There was incontinence of urine and of fæces. The pulse was rapid and irregular, and the extremities were cold. The *tache cérébrale* was obtained for the first time, and lasted for twelve minutes. 0.12 gramme (2 grains) of chloral and 2 grammes (½ drachm) of brandy were given subcutaneously. The convulsions ceased, the opisthotonos disappeared in twenty minutes, and the child remained quiet.

On the 26th the record was that for two days the child had been decidedly better, the retraction and strabismus were less, the nystagmus had disappeared, and he had recognized and spoken to his father. The *tache cérébrale* could be obtained, but was less distinct, and the temperature was normal. On the 28th he became worse again. His head was again retracted, but he was not wholly unconscious. There was retention of urine, for which he had to be catheterized. On the 29th he had a convulsion lasting three minutes, in which the right arm was jerked up over his head. This was followed by partial opisthotonos, and then by a general convulsion lasting two or three minutes, during which his eyes rolled up. At times he would have convulsive movements and tremor without actual convulsions. This condition continued for nine days. He remained unconscious, with his eyes open and staring, the head drawn back, the abdomen retracted, and the *tache cérébrale* very marked. The respirations were of a pronounced Cheyne-Stokes type, and the pulse intermittent. The temperature varied from 37.2° to 38.8° C. (99° to 102° F.), but rose within the last twelve hours to 40° C. (104° F.), indicating that the fatal issue of the case was very near.

On the following day there were a number of convulsions occurring in rapid succession, especially involving the left side. These convulsions continued for five hours before the child died.

The following case illustrates very well the tubercular meningitis of middle childhood:

A boy, five years old, had always been well and strong. On December 3, while endeavoring to climb into bed, he fell and struck the back of his head. He cried afterwards, but the blow left no mark, and nothing was thought of it. The following day, while playing, he fell again and struck the back of his head, but the blow did not appear serious. Two days later he was unable to eat and was somewhat fretful, both of which conditions were unusual for him.

On December 29 he had a slight follicular tonsillitis. His pulse and temperature were normal, the cheeks were flushed, the eyes dull, and the pupils normal. His head was slightly hot, and he was dull and drowsy. His bowels, which had been constipated, on the third day were moved by means of medicine. He continued to be in about the same condition until January 2, when his temperature was 37.2° C. (99° F.), and his pulse 64, regular and strong; his face was flushed, and his eyes were vacant and staring. He vomited once on that night, passed his water involuntarily, moved his left leg spasmodically, and clinched his hands occasionally. He was evidently uneasy, and moaned a good deal. On the following day the pulse was occasionally intermittent. In the mean time he became more and more drowsy, and finally relapsed into a state of unconsciousness.

On the 3d of January the pupils were normal, but he was completely unconscious. The temperature was 38.1° C. (100.6° F.), the pulse 180, and the respirations 30. I saw the child on January 4, and on making a careful physical examination found nothing abnormal, except a slight congestion of the ear in the neighborhood of the malleus, and in the back over the apex of the lung was a slight elevation of pitch on percussion. The temperature was 39.1° C. (101.2° F.), and the pulse was 89 and strong. There was considerable twitching of the arms, chiefly on the right side, lasting from ten to twenty minutes. The pupils were slightly contracted, but were equal. That night he drew his right hand across the face with a quick trembling motion, the right leg being drawn up and the whole body trembling; occasionally there was moaning and sighing respiration.

The examination of the ears showed that there was a slight congestion in the posterior canal of both ears and also in the neighborhood of the right malleus. Both membranæ tympanorum were clear, normal, transparent, and without injection of the manubrial blood-vessels. There was, in fact, no evidence of disturbance of the ears. On the posterior wall of each external auditory canal at the anterior third, more pronounced in the right than in the left ear, was a circumscribed patch of injection such as is observed in cases of inflammatory processes in the mastoid antrum, and occasionally in uncomplicated congestion of the middle ear.

During the next few days the boy's condition varied but little. The eyes, usually closed, would at times open completely, when the eyeballs could be seen to move from side to side. The respiration was sighing, interrupted, occasionally almost inaudible, and then for a time noisy. At times the breathing was suspended for several minutes, when bright red spots would appear on the cheeks; these would disappear when the respiration was resumed. The patient moaned occasionally, and there was some twitching and moving of the extremities, but no convulsions. The pulse was fair in strength, but at times intermittent. The temperature varied, but was moderate in degree.

The extremities of the right side were absolutely motionless, and sensation was apparently absent. The child lay, as a rule, perfectly quiet, as though asleep, and at times would present the picture of a perfectly healthy child sleeping.

On January 9 the extremities became cold, the face very pale, and the pulse imperceptible. This condition lasted fifteen minutes, when he improved in appearance. During the night the breathing grew very rapid, he was restless, moved the left arm continually, and moaned. After some time he opened his eyes, looked around the room, and then became quiet and slept. The next day he was slightly unconscious, and the fingers were flexed, with a very strong contraction of the muscles. The breathing then became more difficult, the nostrils being widely dilated with every breath. During the night he was conscious for some time, swallowed water without difficulty, and the eyes were wide open.

On January 11 there was ptosis of the right eyelid. The pulse became regular, compressible, and intermittent. The left arm was occasionally raised to the head with a quick spasmodic motion, the child moaning as if distressed. Later the eyes became fixed, the pupils dilated, the nostrils expanded, and a bluish color appeared around his mouth and nose. The breathing became very difficult. During an attack of this kind he had every appearance of being moribund, and each attack was thought to be his last.

The change from day to day in the child's general condition was almost imperceptible. He was, however, gradually becoming emaciated.

On January 12 the pupils of both eyes were much dilated; the right eye was almost motionless, with ptosis of the right lid, while the left eye moved occasionally from side to side in a circle. The face was livid, and the hands were mottled with bright red spots. Later, the left eye became quiet and showed a slightly contracted pupil.

On the following day, January 13, the movements of the left eye were repeated, the right pupil being dilated, while the left one was contracted. During all this time the enemata were retained, the bowels moved regularly, and the urine was passed normally. The pulse was so weak that at times it could not be found at all at the wrist, and the breathing was at times inaudible and almost imperceptible.

On January 17 there was slight discharge of pus from the mouth, and also from the left eye. During the next day his breathing grew more and more difficult, and it seemed as though he could not possibly live much longer. In the evening, however, his respiration was much easier and his whole appearance had greatly improved. His breath was very offensive, and there was a loud bubbling sound in the throat.

On January 19 the right nostril was much more dilated during inspiration than the left. The forehead was shiny and slightly œdematous, and the veins were plainly mapped out. Occasionally he moved his right hip-joint and shoulder, which had been motionless for days. There was another slight discharge of pus from the mouth, and when his lips were wiped he seemed more sensitive to touch than before. During the night his left arm and left leg were constantly moved, and he moaned as though he were still in pain. His forehead was still œdematous.

During the next day he was in a state of deep coma for four hours. He then drew a deep sigh and seemed somewhat conscious. The pulse was soft, intermittent and fluctuating.

On January 20 he partly opened and shut his right eye, which was very sensitive to light. The breathing was difficult and noisy. The face was covered with perspiration. At 10 P.M. the sighing respiration began again, and at 10.15 he died quietly, on the thirty-first day of the disease.

The autopsy was made eighteen hours after death and showed tne following pathological conditions: tubercular meningitis, acute hydrocephalus, ependymitis, tuberculosis of the velum interpositum, tubercular nodules in the lungs, tuberculosis of the bronchial lymph-glands, tuberculosis of the spleen, tubercular ulcerations of the intestines.

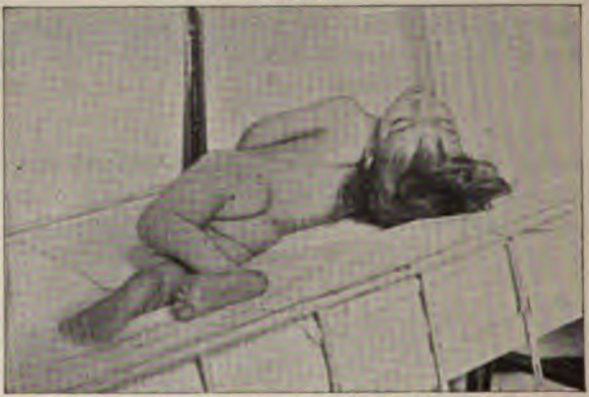
The following case (Fig. 106) simulated cerebro-spinal meningitis very closely, and illustrates an important fact, that a child may recover temporarily from an attack of tubercular meningitis and then die of a recurrent attack.

A child, twenty-one months old, was stated to have been healthy until it was nine months old. At that time it had a convulsion, which first affected the right and then the left side. It was unconscious for ten days, and was somnolent for four weeks. Two or three weeks later its general condition improved. During this time it did not use the muscles of its left side or limbs, and it could laugh only with the right side of its face. Its body was turned continuously to the left; sensation was not interfered with. It gained slowly in strength, and the symptoms gradually disappeared, until it was thirteen months old, when it seemed to be comparatively well, all motor disturbances having ceased. In the following months it had a few slight attacks of the same nature. The final attack from which it died occurred when it was twenty months old, and began with a convulsion on the right side with twitching of the muscles on the left side and frothing at the mouth. There was also ptosis of the left eye. It did not cry out when going into the convulsions, but had marked opisthotonos, which lasted, to a greater or less extent, for five weeks. During these five weeks it was unconscious, and there were several slighter attacks.

On entering the hospital, physical examination showed that the child was of medium size, pale, poorly developed and nourished, and unable to stand, the left leg being weaker than the right. Nothing abnormal was found in connection with the heart or lungs. She could use her extremities partially, but there was an evident motor disturbance of the whole of the left side, and she took hold of objects with her right hand only. The index and little finger of the left hand were frequently found to be extended, the second and third fingers being flexed partially. There was also slight drooping of the left eyelid, and the lines of the left side of the face were obliterated. There was a very slight drooping of the left corner of the mouth. There was slight strabismus of the left eye, and an apparent lack of power of the left external rectus muscle. The patellar reflexes were exaggerated on the left side. Examination showed the ankle-clonus to be absent. The epiphyses of the wrists were somewhat enlarged. The child could not speak, and apparently could not understand readily. No evidence of history of cerebral injury could be obtained. The circumference of the chest was 1 cm. (⅜ inch) larger than that of the head. The cause of the disease was so obscure that at this period the diagnosis could not be definitely made, the supposition being that the child was suffering from the results of an attack of cerebro-spinal meningitis, or possibly from tertiary syphilis, or that a cerebral hemorrhage had taken place, with a resulting spastic paralysis.

While in the hospital the child presented a number of different nervous phenomena. At times she would appear to be for days semi-comatose and would not take notice of anything about her; the eyes rolled up and she would have slight twitching of the body, but this was not localized, and there were no convulsions. At another time, while sleeping quietly during the night, she was found to be unconscious in the morning, and to have her head slightly drawn back and her eyes turned up. Nystagmus was present, and the pupils were dilated and did not react to light, but were equal in size. Clonic twitching of the right foot and the muscles of the right side, flexion of the fingers of the right hand over the thumb, and twitching of the muscles of the wrist sometimes occurred. There was twitching of the fibres of the sterno-mastoid muscle on the right side. There was also twitching of the right side of the face. There was no spasm on the left side, except of the left sterno-mastoid, but there was a nystagmus of the left eye. These clonic twitchings were rhythmical and occurred 180 times a minute. The pulse was 172, and was very feeble. The respirations were 80, rapid and rattling; the temperature was 39.4° C. (103° F.).

FIG. 106.



Recurrent tubercular meningitis. Female, 21 months old.

From 2 A.M. until 5 A.M. 0.36 gramme (6 grains) of chloral was given by enema, and 0.36 gramme (6 grains) of bromide of potassium was given every three-quarters of an hour by the mouth, alternating with the chloral. The spasms became less marked after 3 A.M., but continued in a mild degree up to 11 A.M. During the remainder of the day and the next two days the child lay in a stupor, but had no convulsions. It was able to swallow brandy and milk, which were given to it by the mouth in small quantities at different intervals.

On the next day she apparently had attacks of pain, when she would straighten herself out, throw back her head, and cry out. On the following day, she began to have the same twitchings as in the attack previously mentioned. They were of the same character, except that the extensor muscles of the left foot contracted feebly.

On the following day it was reported that she had had no convulsions, but apparent attacks of pain, when she would cry out and throw her head back, and that she had had another attack of opisthotonos, which was so much more marked than before that the heels almost touched the back of her head. The next symptom which appeared was stupor. The temperature at this time was considerably elevated.

On the following day there were no convulsions, and her condition was about the same as on the previous day, but the head was drawn back and was rigid, and the legs were drawn up and were held rigidly. She lay in this condition, most of the time in a stupor, crying out occasionally, and moving her left hand and arm more than she did the right. At times she would appear to be sleeping naturally and the rigidity would pass away

The opisthotonos gradually become more marked and more frequent in its occurrence, and, although the bowels were moved regularly every day, she took less nourishment, and the temperature continued to rise, and varied from 37.7° to 40° C. (100° to 104° F.).

During the last week of her life the opisthotonos became less marked, and at times passed away entirely. She opened her eyes, but the pupils reacted very slightly. The left pupil became somewhat larger than the right and reacted slightly, while the right pupil did not react at all. The spastic condition of the right wrist and left knee persisted, the patellar reflexes were equal and normal, and the child lay in a semi-stupor, with a temperature varying from 38.3° to 39.4° C. (101° to 103° F.). She took less and less nourishment, and had a slight cough. She gradually lost in weight and strength, and on the day before she died her respirations for a time were very rapid, running up to 100 a minute. Death took place apparently from exhaustion.

The long duration of this last attack, embracing a period of eight or nine weeks, made the diagnosis very difficult.

The autopsy showed the following pathological conditions: subacute tubercular meningitis, chronic granular ependymitis, chronic hydrocephalus, atrophy of the brain-substance, miliary tuberculosis of the lungs, spleen, and kidneys, and chronic tuberculosis of the lung.

Some of the tubercular lesions were of recent growth, while others were evidently old ones and representative of a former attack. The presence of older tubercular lesions in the meninges, as well as of those which produced the symptoms in the last attack from which the infant died, proved that the case was one of recurrent tubercular meningitis.

These cases of recurrent tubercular meningitis are so very rare, as the disease is so uniformly fatal in the first attack, that another case of this kind is given:

A girl, five years old, entered the Good Samaritan Hospital with hip disease on the left side and dorsal Pott's disease. She was treated in bed and did very well for a time, but on May 7, after a week in which she showed anorexia and loss of weight, she began to vomit, and on the following day complained of headache and photophobia. She rolled her head from side to side. Her bowels were constipated, and could not be moved by enemata, and her abdomen was much retracted. This continued for four days, with at times delirium, accompanied by marked drowsiness. There were also ptosis of the left eyelid, slight convulsive movements of the limbs, and frequent movements of the hands to her head, as though she were in pain. On May 12 she had recovered so much that she played with the other children and called for her books and toys. The left pupil, however, remained a little smaller than the right. On the 15th of May, and again on the 20th, 21st, 25th, and 27th, the patient became drowsy and complained of headache. In the intervals between these attacks she seemed bright and well. During the drowsy periods her abdomen was retracted and her bowels were constipated.

From the 27th of May until the 20th of July she appeared as well as usual. On the latter date her temperature suddenly rose to 40.1° C. (104.2° F.). She had pain in the head and photophobia, and the right pupil was larger than the left. This lasted only two days. She then became bright and well again, and continued so for over ten weeks. On October 2, having been perfectly well on the previous day, she began to vomit and to complain of headache. Two days later she fell into a stupor and became completely comatose. On October 6 the left pupil was widely dilated and the right one was contracted to the size of 2 mm. ($\frac{1}{12}$ inch); there were convulsive movements, and later in the day she died.

The post-mortem examination showed a recent tubercular meningitis. In addition to these lesions there were found some older large tubercles of the brain and the remains of the previous attacks of tubercular meningitis. No lesion of importance was detected in the other organs.

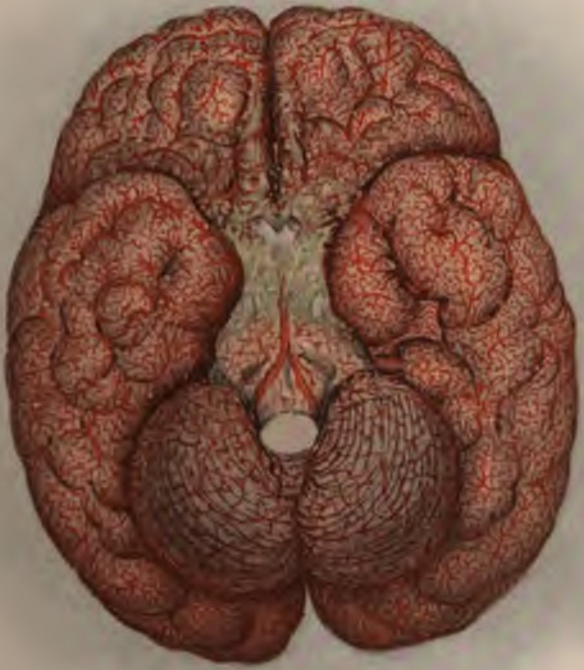
It is not unusual to meet with a tubercular meningitis secondary to tubercular disease of the spine. This complication occurred in the following case in a child four years old:

The child was being treated for Pott's disease with lateral deviation of the spine. He was placed on a frame for five weeks, and at the end of that time he lost in appetite and weight and began to have a cough. Nothing especial, however, was found in the lungs. The bowels became constipated, and he then began to have some mental disturbance and to vomit. A few days later he became unconscious, and on examining him his pupils were found widely dilated, uneven, and not responding to light. His temperature was usually about 38.8° C. (102° F.). The pulse and respirations were somewhat quickened. His head was retracted, and on the day of his death he had a convulsion.

Tubercular meningitis may also occur in connection with disease of the hip, the latter being much more common than when the spine is affected. The following case (8) illustrates this complication:

A child, four years old, was being treated by an irregular practitioner for disease of the hip-joint. The child had been allowed to drag itself about, and the treatment had been with drugs and not by apparatus. When the child was placed under Dr. Brown's care he had him taken to the country and placed in a house and room where all the hygienic surroundings were good. He kept the child in bed and treated it by means of the method of extension usually employed in these cases. The child at first began to improve, but after a few weeks lost in weight and in appetite. Its temperature, which had been varying from 37.2° to 37.7° C. (99° to 100° F.), rose to from 39.4° to 40° C. (103° to 104° F.). A few days later the child became somnolent and had convulsions.

PLATE VI.



Tubercular Meningitis.

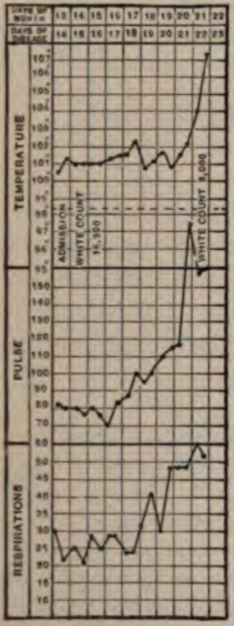
en I saw the case with Dr. Brown it was evidently one of tubercular menin-
arently secondary to disease of the hip-joint, and the child died within twenty-
rs after I had examined it.

: tubercular form of otitis is not uncommon, and it may be the
-point for tubercular meningitis. Surgeons should, therefore,
carefully the possible complication of tubercular meningitis when
tubercular disease of the bones and joints and of the ear.

: following case was one of tubercular meningitis, associated with
te miliary tuberculosis, occurring in my wards at the Children's
l. The pathological appearances on the base of the brain, as
on autopsy, are represented in Plate I., frontispiece.

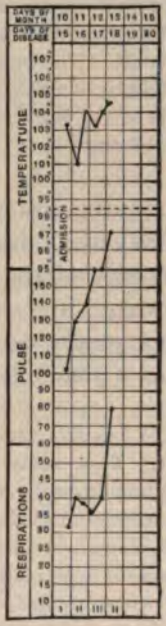
: child entered the hospital on September 13. She was three years of age.
e exception of an attack of whooping-cough she had been well until two weeks
ntrance, when she was taken with vomiting and diarrhœa. She complained of
e and pain in her stomach, and there had been continuous fever. The diarrhœa
few days, and she then became constipated. Four days before entrance the
of her neck became stiff. No further history could be obtained.

CHART 9.



ubercular meningitis. Age, 3 years.

CHART 10.



Tubercular meningitis. Age, 2 years.

e physical examination showed the child to be well developed and fairly nour-
ut anæmic. She lay in a stupor most of the time, but could be aroused. The
ere equal and reacted sluggishly. The tongue was coated, the throat, heart, and
owed nothing. The head was slightly retracted, and the posterior muscles of
 were rigid. The abdomen was not retracted, and nothing abnormal was de-
A *tache cérébrale* was present. The knee-jerks were normal. Babinsky's
s present in both feet, and Kernig's sign was also obtained.

On September 14 there were some convulsive movements of the right extremities, and breathing was somewhat labored.

On September 15 the child took her food very poorly, and could not swallow well. There were convulsive movements, but no general convulsion.

On September 16 the legs were rigid and held in extreme extension most of the time. The pupils were unequal and dilated, and did not react to light.

On September 17 strabismus of left eye developed, but was not constant. There was convulsive twitching of the limbs, with rigidity most of the time. The face was flushed, perspiration was very free, and the abdomen was somewhat distended.

On September 18 the condition remained the same, except for the additional symptom of Cheyne-Stokes respiration. The rectal feedings were not retained, and it was impossible to insert a catheter for nasal feedings, owing to the swollen condition of the mucous membrane of the nares and the difficult breathing.

On September 21 the respiration had become more sighing in character. The child perspired very freely. The white blood count was 8000.

On September 22 the condition, which had remained practically the same, except for the rising temperature and accelerated pulse, became worse. The child seemed to grow weak rapidly, and the respirations and pulse became irregular and the child finally died. Chart 9, page 425, shows the course of the temperature.

Lumbar puncture was performed immediately after death, and a slightly cloudy fluid with flecks of fibrin was obtained.

The autopsy showed the original focus to be in a bronchial lymph-gland, which was enlarged and caseous, with a cavity at the centre. There were found miliary tuberculosis of the lungs, liver, spleen, and kidneys, tubercular meningitis, and tubercular bronchial glands. The brain weighed 1080 grammes. At the base, around the pons, and in the fissure of Sylvius there was a yellow, gelatinous material, while along the blood-vessels of the pia were scattered numerous miliary tubercles, sometimes single and again confluent.

The following was a case of tubercular meningitis with symptoms of comparatively short duration and associated with a general miliary tuberculosis. It is especially interesting in that the tubercular process, as shown by the autopsy, and as represented in Plate I., frontispiece, was most highly and typically developed on the convexity of the brain instead of at the base, as is generally the rule.

The child was two years old. She had been losing flesh and strength for about two months. Two weeks before entering my hospital wards she was taken with vomiting without special reference to the taking of food. This was the only symptom the parents noticed until three days before entrance, when convulsions and fever developed. Nothing more definite could be learned from the parents.

The physical examination showed a child who was fairly developed and nourished, pale, pupils widely dilated, the right reacting sluggishly, and the left not at all. The eyes were rolled up, but there was no strabismus. The head was retracted, there was no tenderness along the spine. The tongue was slightly coated. There was a slight rosary. The throat, heart, lungs, and abdomen presented nothing abnormal so far as could be determined. There were no petechiæ. A *tache cérébrale* was present. The knee-jerks were slightly exaggerated on both sides. The plantar reflexes were normal. Babinsky's sign was present in both feet. Kernig's sign was absent. The child lay in a stupor. The white blood count was 6000.

On the following day there were convulsive twitchings of the right arm and right side of the face. On the third day after entrance these signs became more marked, the respirations Cheyne-Stokes in character, the pulse rapid, irregular, and small. There were no convulsions of the entire body, but the right arm was flexed at the elbow. These symptoms continued until the fourth day, when the child died. The course of the temperature is shown in Chart 10, page 425.

Lumbar puncture, done immediately after death, gave a slightly turbid fluid, and flecks of fibrin separated out. No tubercle bacilli were discovered in the sediment from lumbar puncture.

At the autopsy the following condition of the brain was found :

The dura was tense and bulging ; the superior longitudinal sinus was normal ; the gyri were flattened ; the sulci were partly obliterated ; the vessels of the pi-arachnoid over the vertex were markedly injected, and the cerebro-spinal fluid considerably increased. In the pia mater over the superior surface of the right frontal lobe, and extending to the mesial surface, was an area 5 by 3 cm., with opaque, grayish-yellow, single and conglomerate tubercles ; the single tubercles averaging one mm. in size. Tubercles were present in great numbers in the sulci, adjacent to the blood-vessels, but were also distributed upon the gyri over both hemispheres, but especially over the lateral surfaces of the parietal lobes. About the optic chiasm and interpeduncular spaces was a mass of semi-opaque gelatinous material beneath the turbid pia. Around the branches of the middle meningeal artery, and especially in the Sylvian fissure, was an abundant diffuse fibrino-purulent exudation. Scattered tubercles were found over the base, but in less numbers than over the vertex. Many were scarcely visible, being nearly transparent and less than a millimetre in diameter.

The anatomical diagnosis was : tubercular meningitis, acute miliary tuberculosis of the lungs, tubercular ulceration of the intestines, tuberculosis of the mesenteric lymph-nodes, and a mild degree of rhachitis.

TUBERCULOSIS OF THE JOINTS.

Tubercular disease of the joints is essentially a disease of childhood ; it is rarely congenital, and under one year is not common. In 1344 cases of hip disease, 1000 occurred under fifteen years of age. (Bradford and Lovett.) In hip disease, for example, a series of cases from the New York Dispensary showed that 115 occurred under three, 316 from three to five, 509 from five to ten, 140 from ten to fifteen, and 98 over fifteen years. This may be taken as fairly representative of the general liability to the diseases at different ages.

At the Children's Hospital, Boston, from 1869 to 1893, 3820 cases of tuberculosis of the joints were as follows :

TABLE 62.

Spine	1964
Hip	1402
Ankle	300
Knee	104
Wrist	20
Shoulder	15
Elbow	15

These figures are similar to those reported by other American authors.

With regard to the causation of tubercular disease of the joint, it may be stated that the element of heredity is to be found in a large proportion of cases, which is variously estimated, the percentage, according to different authors, varying from twenty-four (Croft) to sixty-eight (Gibney); an average estimate would perhaps be forty per cent. (Nichols).

Traumatism is accepted as being the cause of chronic disease of the joints in certain cases (König); this percentage, also, is variously estimated from thirty-five to fifty per cent. Chronic tubercular disease of the joints follows in certain cases after the exanthemata, especially measles and scarlet fever.

Pathology.—Tuberculosis of the joints in children, as well as in adults, is now much more uniformly regarded as being of bony origin than was formerly supposed.

The existence of a primary synovial tuberculosis in children must be regarded as extremely rare, and only to be demonstrated by the examination of all parts of the bones entering into the formation of the joint. Finding a tuberculosis of the synovial membrane at operation, with an obvious bony lesion, cannot be accepted as proof of primary synovial disease.

The original statement of Volkmann is, "The fungous inflammation of the joints in children and adults begins generally not at all as an arthropathy, but as a pure osteopathy, with a very circumscribed condition of tubercular osteitis."

Nichols, in the examination of one hundred and twenty tubercular joints from children and adults, did not find one joint where, if all the bones entering into the joints were sawed open in thin layers, one or more old bone foci were not found.

The process by which joints become affected by tubercular disease is as follows: A tubercular focus develops in the spongy tissue of the bone near the epiphysis, and by an extension of this process the bone is destroyed in the line of least resistance, which is usually towards the surface of the joint. When perforation of the joint by this focus occurs, the tubercle bacilli obtain access to the joint cavity and a tubercular synovitis begins. From this point destruction in any direction is, of course, possible. Under favorable conditions of rest and separation of the surfaces of the joint by traction. in certain cases, nature tends to limit the process by repairing and replacing the tubercular tissue by the formation of fibrous tissue which grows into and replaces the tubercular material. The soft parts around the joint are, of course, secondarily affected, and abscesses are a frequent complication.

The contents of a tubercular abscess may be sterile so far as pyogenic organisms are concerned, or they may contain, especially in their later stages, pyogenic organisms. Tubercle bacilli are to be found in these abscesses in only about one-third of the cases, and, as a rule, only after prolonged search. Inoculation experiments must be relied upon in the majority of cases to establish their presence. The presence of leucocytosis in a child with disease of a joint does not establish the presence of pyogenic organisms

Symptoms.—Hip.—Chronic tubercular disease of the hip-joint, commonly known as hip disease, most often begins with lameness, paroxysmal cries at night, and a stiffness in the joint which is more noticeable in the morning than at night. The pain is more often referred to the knee than to the hip, and may be very severe or may be slight.

On examination in the early stages, the joint is found to be somewhat limited in its motions, the trochanter is slightly thicker than the one in the well limb, and wasting of the muscles occurs in the thigh and the calf.

The prognosis in early cases is excellent for the restoration of a useful limb, and even in the late cases the adoption of treatment by traction generally brings about good results. It must, however, be remembered that these children are tubercular, and that a certain number of them develop tubercular meningitis in the course of the disease.

Spine.—Chronic tubercular disease of the spine, or **Pott's disease**, begins usually with stiffness of the back and pain on exertion, which is in most cases referred to the peripheral end of the nerves, so that the pain may be complained of either in the abdomen or chest. Such children walk with a guarded gait and stiffly, and when tired support themselves by leaning on furniture or by resting the hands upon the thighs.

In the cervical region torticollis and difficult breathing at night may be the only symptoms. In the lumbar region one symptom generally noticed is an apparent lordosis, in which the child leans far backward. Paralysis, which occurs in a certain proportion of cases due to a pressure from meningitis originating at the seat of the disease, may be the first symptom; it is accompanied by increased reflexes and a loss of power in the legs. Psoas abscess is a common complication in the lower part of the column, and the first symptom noticed in this complication is generally inability to hyperextend the leg upon the affected side. The chief sign to be relied upon in the diagnosis is stiffness in some part of the spine. This is most easily detected by having the child stoop to pick up objects from the floor, and by laying the child on the face and flexing the spine by lifting the body by the legs. The appearance of a knuckle occurs early in the disease, but it is most important that the diagnosis should be made before this knuckle occurs, or while it is still small.

The prognosis is perhaps not so favorable as in hip disease, but in early cases the results are usually excellent, and even in late cases much is to be gained from accurate support to the spine.

Knee.—Chronic tubercular disease of the knee-joint, or *tumor albus*, is most often made evident by swelling of the knee of a chronic character, as a rule accompanied by pain. The child is apt to walk with the knee somewhat bent, and it is often difficult to fully extend it. Motion in the knee-joint is limited and the joint may or may not contain fluid. The character of the swelling is commonly that of a gelatinous mass involving the joint rather than a mass of fluid filling it. The occurrence of bony enlargement is most significant as probably establishing the existence of chronic tuberculosis.

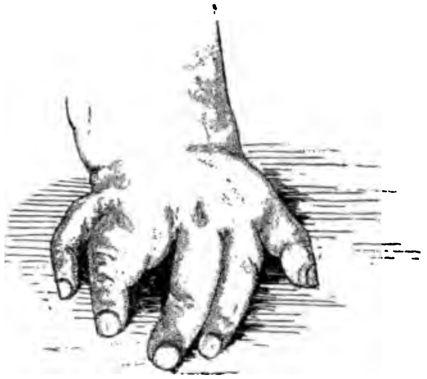
In the diagnosis much difficulty is often experienced in determining between chronic disease and sprains. It may be said in general that chronic sprains in children are not very common, and that it is not wise to make such a diagnosis until after recovery has occurred.

As to the prognosis, chronic tuberculosis of the knee-joint apparently runs a much less severe course than that of the hip or spine, and is most favorably affected by efficient treatment.

Chronic tubercular disease of the other joints is characterized by swelling, more or less stiffness and pain, and such joints, as a rule, present the same symptoms as those described in speaking of the knee.

Tubercular Dactylitis.—Tubercular dactylitis is a tubercular disease of the phalanges of the hand and foot. Usually, the cartilaginous ends of the bones are not affected; the bone appears larger in diameter than is normal and is pyriform in shape. This enlargement is due to the increased size of the periosteum which forms a new layer of bone, and this new layer of bone may be very thin. Frequently the tubercular centre opens to the surface by one or several tubercular sinuses; healing in such cases may occur spontaneously, but operation in advanced cases is advisable. A permanent deformity may arise from the removal of the diseased phalanx.

FIG. 107.



Tubercular dactylitis.

Fig. 107 represents a tubercular dactylitis of the third finger of the left hand of an infant.

TUBERCULOSIS OF THE THYROID GLAND.

Although tuberculosis of the thyroid gland may occur as a primary disease, it is exceedingly rare, the disease practically being always secondary to a general miliary tuberculosis, and not presenting symptoms which, as a rule, can enable us to make a diagnosis during life. Amyloid degeneration of the thyroid gland may occur as the result of a chronic tuberculosis, especially of the joints.

TUBERCULOSIS OF THE THYMUS GLAND.

Tuberculosis of the thymus gland may occur in general tuberculosis, but is never primary.

TUBERCULOSIS OF THE PANCREAS.

Tuberculosis of the pancreas may occur in general tuberculosis, but is never a primary disease. Amyloid degeneration of the pancreas may occur as a result of chronic tuberculosis, especially of the joints.

TUBERCULOSIS OF THE SPLEEN.

Tubercular lesions of the spleen are always secondary. They occur only in the form of small miliary tubercles. Amyloid degeneration of the spleen may occur as a result of chronic tuberculosis, especially of the joints.

TUBERCULOSIS OF THE LIVER.

Tubercular lesions of the liver are always secondary. They appear in the form of small miliary tubercles and cheesy nodules. Amyloid degeneration of the liver occurs in certain cases of tuberculosis, especially where there is wasting. Tuberculosis is not, however, a primary disease of the liver, but a symptom of tuberculosis elsewhere, especially of the bones and joints, and is not so common in children as in adults. Tuberculosis of the liver occurs in connection with general tubercular disease of other organs, and does not in itself present any especial characteristic clinical manifestations. The rare instances in which large caseous masses cause obstruction and, later, disintegration of the tissues, with hepatic enlargement and abscess, are not usually recognized during life.

TUBERCULOSIS OF THE KIDNEY.

Tuberculosis of the kidney in children occurs in two forms: (1) As a miliary tuberculosis, the tubercle bacillus being conveyed into the tissues by the blood. This form rarely reaches any clinical importance. (2) As a tuberculosis beginning in the pelvis of the kidney and extending from this into the cortex. This form is by far the most serious in its symptoms and results. The detection of the tubercle bacillus in the urine in these cases is the only positive sign of the disease. The symptoms are those of a pyelonephritis represented by pus in the urine, an irritable bladder, and sometimes the presence of a tumor or abscess in the renal region. The treatment is essentially surgical. Amyloid degeneration of the kidney may occur as a result of chronic tuberculosis, especially of the joints.

TUBERCULOSIS OF THE BLADDER.

Tuberculosis of the bladder is very rare.

TUBERCULOSIS OF THE TESTICLE.

As compared with the frequency of its occurrence in adults, tubercular disease of the testicle is rare in infancy and childhood. When the disease is present the gland is considerably swollen and often nodular, but rarely very tender. As the disease progresses adhesions may form with the tissues of the scrotum, and the degenerated material may be discharged through a fistulous tract. General treatment is indicated if the disease is just starting, but if it has already destroyed the usefulness of the gland it is safer to operate immediately and remove the focus of infection. Here, of course, we should be guided by the conditions elsewhere.

TUBERCULOSIS OF THE SKIN.

The cause of what is now known to be tuberculosis of the skin (lupus or scrofuloderma) was for many years unknown. It was designated by various terms according to the different forms which it assumed on the skin. Thus, in one form it was called *lupus*, and in another *scrofula*. We now know that all these forms are caused by the same micro-organism, the tubercle bacillus, and that this bacillus may find its nidus in the skin, as it does in all other organs of the body,—that is, we may have a local tuberculosis of the skin. The disease does not differ in the child in its appearance, its course, and its general symptoms from that met with in the adult. As a rule, it causes, next to syphilis, the greatest destruction of tissue of any known disease of the skin. The time of its appearance varies, but it is more common in adults than in children.

Treatment.—The treatment is the same as in adults. The fundamental object to be obtained is the destruction of the diseased tissue. Where there is a small isolated area which can be easily removed by the knife, this method of treatment should be employed. We must remember, however, that by this method it is almost impossible to avoid removing the sound tissue with the diseased, and that such good results as the avoidance of unsightly scars are not obtained so well by this method as by others. Therefore, when the tissues are extensively diseased, and areas are involved where it is desirable to avoid scarring, such as the face, a locality which is very frequently attacked by tuberculosis, the actual cautery and electro-cautery may preferably be used. Various caustics, as the solid stick of nitrate of silver, as recommended by the Vienna School, have been found of much value. This disease, under all circumstances, is very intractable to treatment; and often causes great deformity.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

Epidemic cerebro-spinal meningitis is a primary acute infectious disease caused by the diplococcus intracellularis, and characterized primarily by lesions of the piarachnoid of the brain and cord.

Etiology.—The cases of cerebro-spinal meningitis which have occurred in Boston and its vicinity, including my own cases at the Children's Hospital, reported by me in 1895, have been so exhaustively treated by Councilman, and Mallory and Wright in the Report of the State Board of Health of Massachusetts for 1898, that in my description of the disease I must acknowledge at once my indebtedness to these investigators for the use which I have necessarily made of their work.

As, for the first time in the history of the disease, the lesions have been studied from the stand-point of their bacteriology, this work should be taken as the standard by which we are to be guided in the study of the pathology, symptoms, diagnosis, and prognosis of the disease.

Cerebro-spinal meningitis was first recognized and described by Vieusseaux in Geneva in 1805. The first case which occurred in America was in 1809. The disease may occur in beasts as well as in man, and has been much more wide-spread and frequent in America than in Europe. It is rare in infancy, but is more common in children than in adults. The contagious nature of the disease is somewhat doubtful. There seems to be a high degree of immunity to a second attack. The incubation is unknown. The disease may be epidemic or sporadic. The mode of entrance of the organism is unknown, but may possibly be through the nose.

PATHOLOGY.—*Nervous System; Meninges.*—The inflammatory process is characterized by purulent, sero-purulent, and fibrino-purulent exudation, chiefly in the sulci along the vessels of the choroid plexus, and is usually most pronounced at the base, but at times is more marked on the convexity of the brain, especially over the parietal and occipital lobes. The meninges of the cerebellum are always affected. The meninges of the entire brain are rarely involved, although occasionally large areas are included in the inflammatory process. The longer the duration of the disease the more extensive are the lesions. In the very chronic cases œdema and general thickening of the meninges are the characteristic features of the lesions.

The inflammation is confined chiefly to the piarachnoid; the dura is but slightly involved, and the subdural space is increased by the fluid exudate.

In the most acute cases, in which the macroscopic changes are very slight, the lesions are shown by microscopic examination to consist chiefly of purulent infiltration of the meninges, with injection of the blood-vessels. The polynuclear leucocytes are massed in some places, scattered in others, and with but little fibrin among them. The absence of eosinophilic cells is notable. There is no evidence of proliferation of tissue.

In the more advanced cases, on the other hand, there are large numbers of cells, chiefly polynuclear leucocytes, lying in the dilated lymph-spaces of the tissue. The presence of red blood-corpuscles is rare. The fibrin is present in considerable amount, often showing hyaline changes, but is never so great as in the meningitis produced by the pneumococcus. Large epithelioid cells, from two to eight times the diameter of a leucocyte, and filled with leucocytes, or rarely with lymphoid cells, are often seen, but their origin is obscure. The vessels are dilated, and at times contain thrombi. Proliferative changes within the intima of the arteries, which are common in tubercular or pneumococcus meningitis, are rarely found.

In the acute cases the macroscopic and microscopic changes in the meninges of the cord are very similar to those of the brain. The cord is always somewhat affected, and occasionally to a greater degree than the brain. The injection of the dura is especially intense. The fluid in grayish foci with softened centres and hemorrhagic and œdematous tissue between them are found. The central areas represent a purulent infiltration of the tissue with abscess formation. The foci of consolidation are not bronchial in origin. The infection of the lungs by the diplococcus is probably by means of the blood rather than of the bronchi, and may occur in almost any period of the disease. In all these cases immense numbers of diplococci are found within the pus-cells.

Spleen.—The size of the spleen is generally smaller than in most of the acute diseases, although there is considerable variation. The increase in size is due chiefly to hyperæmia. There is very little change in the histological appearances of the spleen.

Lymphatic Nodes.—There is usually no enlargement of the lymphatic nodes except in the complicated cases, and there are no microscopic changes beyond the injection of the blood-vessels.

Liver.—The liver is usually pale and cloudy on section, and in some cases there is slight increase in the fibrous tissue with cellular infiltration.

Kidneys.—The kidneys show acute, degenerative lesions in all cases, but a nephritis is rare if the meningitis is uncomplicated.

Skin.—Lesions of the skin are rare; when present, they are in the nature of ecchymoses with intense congestion of the surrounding vessels, and with some infiltration with pus-cells beneath the epithelium.

In addition to the parenchymatous degeneration of the heart, liver, and kidneys, there are a certain number of other secondary lesions frequently met with, such as subserous, punctate hemorrhages in the endocardium, hyaline and granular degeneration in the voluntary striated muscles, occasionally multiple abscesses in various parts of the body, suppurative inflammation of the joints, and choroiditis.

The lesions are essentially the same in the epidemic and sporadic cases of cerebro-spinal meningitis.

TYPES OF THE DISEASE.—There are certain types of epidemic cerebrospinal meningitis which differ so markedly as to deserve special mention. The cases may be either *acute* or *chronic*. The acute cases are those in which active symptoms last for about fifteen days, and two other forms may be recognized,—the mild and the fulminating.

Mild Form.—The mild form occurs more frequently in children than in adults. It has an acute onset, short course of one or two days, and rapid subsidence. These cases are sometimes called "abortive," but have not been verified as yet by lumbar puncture.

Fulminating Form.—This term is restricted by Councilman to those cases which are fatal within forty-eight hours from the onset of the disease.

Chronic Cases.—The chronic cases are those represented by less active onset, by remissions and exacerbations, and long duration.

Intermittent Forms.—These cases are chronic in type, and are seen in those instances in which, with or without abatement of the other symptoms, there are complete intermissions in the temperature.

SYMPTOMS.—The chief symptoms are those connected with the nervous system, such as vomiting, delirium, coma, paralysis, pain, retraction of the neck, opisthotonos, and the mental condition after the attack; also those symptoms connected with the lung, with the skin, the eye, the ear, the nose, the throat, and the joints; also the pulse and temperature, and the condition of the blood and spinal fluid. In some cases the onset is not so sudden, and difficulties have arisen in the differentiation from typhoid fever and pneumonia; but, except in the rather rare meningeal types of these latter diseases, the diagnosis will in a few days become clear.

In young infants the symptoms of cerebro-spinal meningitis may be merely a heightened temperature with clonic convulsions, so that the diagnosis cannot be made during life from the various forms of reflex convulsions which may occur at this age, and cerebro-spinal meningitis can only be suspected. General infection by the diplococcus intracellularis is unknown. The mild cases may subside in a few days; the acute cases may last for a number of weeks, and are often fatal. Some cases are so very sudden and violent in their development that they resemble a general toxic infection, and rapidly prove fatal in even a few hours (*foudroyant*). Some cases show decided intermissions in intervals of several days, during which the child seems much better, the temperature is normal or slightly raised, and the pain and tenderness are much diminished. The symptoms then return. Marked emaciation occurs in these cases. In some cases after the usual acute onset, an intermission may come, of a much longer interval of weeks, as to the severe symptoms and the fever; but the other symptoms are apt to continue, and the disease may assume a chronic form, lasting three or four months.

Vomiting.—Vomiting is a very common symptom, and may from its constancy become a dangerous one. It may occur early or late in the disease, and is due to direct or reflex irritation of the vomiting centre, being regarded as of cerebral origin.

Delirium.—Delirium is a common symptom; it may occur early or late in the disease, may be violent or moderate, continuous or intermittent, not more frequently present in the fatal cases than in those which recover, and does not always correspond to an increase of temperature or to an aggravation of the other symptoms.

Pain.—Pain, especially in the head, is a very common symptom, and is at times violent. It may be located either in the occipital or frontal regions, and may extend down the back. In some cases, especially in children, there are abdominal pains and also pains in the extremities. An almost constant symptom is hyperæsthesia of the skin; the patients do not like to be touched, and often the least jarring of the bed causes intense pain. According to Councilman, the general pain in the head is due to the inflammation of the meninges. The pain in the cervical region and back may be referred to pressure exerted on or inflammation of the posterior nerve-roots.

Coma.—There are great variations in the cerebral symptoms: there may be stupor, drowsiness, or deep coma, varying from day to day or from hour to hour; insensibility may suddenly change to consciousness. Coma may occur in the beginning, and the comatose condition may continue until death.

Neck and Spine.—Symptoms connected with the neck are almost always present, and vary from a simple stiffness to rigidity of various degrees, with or without retraction. Attempts to move the head are usually painful. The retraction of the neck is sometimes accompanied by opisthotonos, and all these symptoms can be referred to the effect of pressure on or inflammation of the spinal nerve-roots.

Paralysis.—According to Bullard, these cases, while at times and in certain stages precisely resembling cases of paralysis occurring in anterior poliomyelitis, are to be distinguished clinically from the paralyses of this disease by the following conditions:

1. In cases of paralysis following cerebro-spinal meningitis we find that pain on passive motion of the limbs persists to a degree rarely, if ever, found in anterior poliomyelitis. Such pain and tenderness sometimes exist during the acute stage of anterior poliomyelitis for two or three days, but if they last longer than a week the case is one in which the diagnosis is to be very carefully considered. It is not uncommon, on the other hand, for great pain on passive motion of the limbs or of certain joints, especially of the ankles, to exist in cerebro-spinal meningitis for one, two, or more months after the acute stage of the disease has ceased. Tenderness on pressure over the muscles also persists much longer than in anterior poliomyelitis, where it rarely continues much beyond the acute stage, but does not last as long as the pain on motion, active or passive.

2. The character of the paralysis also differs, at least in the early stages, although later it is often nearly indistinguishable. There is always a tendency to spastic contracture in the early stages of the paralysis of cerebro-spinal meningitis. This is sometimes so marked that it is evident on casual examination. Certain portions of the limbs are flexed, and there is a decided muscular resistance to passive motion due to a permanent tonic contraction. More often, and particularly in the later stages, this tonic or spastic condition is not so evident, and shows itself only or principally in the extreme degree of flexion at the ankle-joint, the foot often being in a position of equinus, and its axis almost in direct continuance with that of the leg.

3. The knee-jerk is usually less affected than in anterior poliomyelitis. In other words, there seems to be a greater tendency to retain the knee-jerk. This is rather a question of degree than of anything else, and may have been accidental in Bullard's patients; he lays stress on it only in connection with other symptoms. The knee-jerk is sometimes in this affection, as in anterior poliomyelitis, totally abolished.

4. In addition to these clinical signs we have the history of the initial

attack to guide us. While certain cases occur in which it is difficult to determine the character of the initial attack, as a rule, the existence of retraction of the head and of contractures of the muscles of the back or limbs, the continued presence of extreme tenderness or pain on movement, and the duration of the acute stage of the disease, enable us to determine more or less accurately the character of the disease.

Any case is suspicious in which the acute stage lasts more than seven days.

Lungs.—Although instances of pneumococcus pneumonia are quite often met with in connection with meningitis, and sometimes with diplococcus meningitis, yet it will probably be found in the future that the meningitis which accompanies pneumococcus pneumonia is a pneumococcus meningitis, while in the epidemic meningitis the complicating pneumonia is caused by the diplococcus intracellularis. This view is especially upheld by Leichtenstern, who states that while epidemic meningitis is rare in many countries, occurs chiefly in early life, and has no crisis, pneumonia is common all over the world, at all ages, and has a distinct crisis. The complications of the two diseases are different. Multiple synovitis and affections of the eye, so frequent in epidemic meningitis, are rare in pneumococcus meningitis. The exudation is more fibrinous in the latter disease, and the result usually rapidly fatal, while the epidemic form is frequently recovered from. The remissions and exacerbations, varying course, relapses, and uncertain gait belong exclusively to the epidemic form. Leichtenstern also believes that the cerebral symptoms dependent on a pneumococcus pneumonia subside at the time of the crisis, just as the other symptoms do. The diplococcus intracellularis can enter the lung and produce a focal pneumonia characterized by especial anatomical features, and occurring in both the acute and chronic cases.

Skin.—The lesions of the skin gave, in the earlier epidemics, the name of spotted fever to the disease. These lesions vary greatly, and may be represented by petechiæ, larger hemorrhagic foci, or circumscribed areas of hyperæmia; the most common form of efflorescence is, however, herpes. In numbers of cases there are no lesions of the skin whatever. A *tache cérébrale* is of common occurrence.

Eye.—Symptoms connected with the eye are prominent in epidemics of meningitis, and there is a marked tendency for the infection to extend along the optic nerve, as already described in the pathology. The cause of the lesions (neuritis or degeneration of the nerves of the eye) may be due to their involvement in the exudation at the base of the brain without any extension of the inflammatory process to either the orbit or the eye. Or the optic nerves may also be involved in this exudation. Again, the inflammation from the meninges may extend directly from the brain into the eye by means of the piarachnoid. All of the cases of purulent choroido-iritis and the very rare cases of suppuration in the orbit are probably due to such extension. There is no metastasis in these conditions, but a direct extension. A third cause of the lesions of the eye, and one to which most of the cases of keratitis are due, is a neuritis of the fifth nerve, with destruction of the Gasserian ganglion and a loss of sensation.

The pupils are generally altered. Reaction to light is slow and incomplete, and dilatation is very apt to occur. Paralysis of the ocular muscles with resulting strabismus may develop, but the paralysis is often temporary, and different muscles may be affected at different times.

Ear.—As in the eye, so in the ear, the infection may extend along the auditory nerve. There may be pain, tenderness of the mastoid, and deafness with or without otitis media. The lesions in epidemic meningitis are always secondary and are due to extension from the brain, while when the meningitis is secondary to an otitis media the organism of infection is the pneumococcus or streptococcus. These lesions of the ear are the most common complications of epidemic meningitis, and in a large number of cases deafness results.

Nose and Throat.—Acute coryza and inflammation of the throat may occur. The lesions may be due to a primary extension from the brain or from a direct secondary infection from the ear. These questions, however, have not yet been satisfactorily determined, and it is possible that the diplococcus intracellularis may be found in the nasal secretion of patients without meningitis.

Joints.—Acute inflammation of the joints is frequent in epidemic meningitis. The knees are the joints most commonly affected.

Pulse and Temperature.—A marked characteristic of epidemic cerebrospinal meningitis is the inequality of the pulse and temperature. The pulse may often remain normal while the temperature is heightened, and there is no relation between the height of the temperature and the severity of the symptoms. The temperature rarely reaches 40° C. (104° F.), and varies from 39° C. (102.2° F.) to 38° C. (100.4° F.). The type is often remittent. The fever gradually disappears and the temperature curve is interrupted by irregular rises and falls. A terminal rise of temperature occurs in some cases. The pulse shows the same irregularity as the temperature, and may range from 70 to 130 in a minute. Relative slowness of the pulse to the temperature is often found in children. Absolute slowness of the pulse is not common.

Blood.—Leucocytosis is always present. The leucocytes gradually diminish towards the end of the disease in the cases which recover.

Spinal Fluid.—In cerebro-spinal meningitis the character of the fluid is more or less turbid, and in severe acute cases a puriform deposit frequently settles to the bottom of the test-tube in a short time; there are numerous polymorphonuclear leucocytes (pus-corpuscles) and occasional smaller mononuclear lymphoid cells and fibrin. Groups of the diplococcus intracellularis are found in varying numbers in the protoplasm of some of the leucocytes. The fluid should be withdrawn at the time when the active symptoms are present, or the growth of the organism may fail to be obtained. A microscopic examination is necessary in all cases, as the macroscopic appearance of the fluid is not to be relied on. Inoculation experiments are, as a rule, unsuccessful, as the organism is not pathogenic for guinea-pigs. Goats are susceptible to it.

Mental Condition after Recovery.—In some cases after recovery from the acute symptoms a condition of mental impairment remains.

DIAGNOSIS.—The differential diagnosis of cerebro-spinal meningitis is essentially from other forms of meningitis, whether tubercular or non-tubercular. The sudden onset, extreme headache, hyperæsthesia, opisthotonos, rigidity and retraction of the neck, pain and tenderness of the neck and spine, herpes, moderate temperature in comparison with the severity of the symptoms, and rapid development of apathy and coma in keeping with the early exudation of pus over the surface of the brain, are in strong contrast to the slow onset and gradual development of the symptoms in tubercular meningitis, described on page 407. The non-tubercular forms of acute meningitis are more difficult to differentiate, and the only exact means of diagnosis is by lumbar puncture. Epidemic influenza at times simulates epidemic cerebro-spinal meningitis so closely that the differential diagnosis can only be made by spinal puncture.

PROGNOSIS.—The prognosis, where the child is young and the onset is violent, with high temperature and continuous convulsions, is very serious, as the disease is one of the most fatal in childhood, and only about one-half recover (Sachs); but even in the apparently fatal cases, where coma has intervened, a change may take place and the child recover. The first two weeks are usually the critical period, so far as the acute form of the disease is concerned. According to Sachs, the cases in which the coma develops rapidly, and does not show any signs of receding within the first week or two, are almost certain to end fatally. If the coma has been slow to develop, the process is usually less intense and the prognosis is better; but even in these cases, if the coma remains stationary for a week or more the chances of recovery lessen every hour that the coma continues.

The chronic form is apt to prove fatal, both from exhaustion and from the development of more serious central nervous lesions.

The cases which recover are often left with permanent lesions, causing deafness, blindness, various paralyses, and mental impairment. Death is very apt to occur quietly.

TREATMENT.—The treatment of cerebro-spinal meningitis varies according to the severity of the symptoms. In most cases sedatives, such as the bromides, are indicated, and where the pain is severe opium in considerable doses is often needed. The ice-bag or Leiter's coil applied to the head, and absolute quiet in a darkened room, are important adjuvants to the treatment. In many cases the pulse becomes so weak and the prostration so marked that stimulants are required until convalescence is established, when they can usually be replaced by tonics. In some cases the hyperæsthesia and general sensitiveness to noise, light, and motion in the room are so extreme and so characteristic that the attendants should be cautioned not to touch the child or the bed unnecessarily, and absolute quiet should be enforced in the room and throughout the house.

The following cases illustrate cerebro-spinal meningitis; where, as in some instances, the diplococcus intracellularis is not mentioned, it is because these cases occurred before lumbar puncture was commonly used for diagnosis.

A boy, thirteen years old, had never had any special diseases, but had been rather delicate for a number of months. He went to a Christmas party on December 25, and on returning from the party complained of the motion of the sleigh in which he rode home. On the following day, in the afternoon, he was found to be listless, to have his tongue coated but not dry, a temperature of 40.5° C. (105° F.), and a pulse of 140. He complained of tenderness and pain in the back of his neck; there was also tenderness in the abdomen. He appeared to be somewhat dull.

On the following day the temperature in the morning was 39.4° C. (103° F.), and the pulse was 120. He was much more dull and apathetic than on the previous day, and in the afternoon became delirious. In the evening he had involuntary passages of urine and loose discharges from the bowels. His temperature was 40° C. (104° F.).

On the following day his temperature was 39.4° C. (103° F.), and the respirations varied from 40 to 80 and were regular. He was unconscious. Subsultus tendinum was present. There was retraction of the head. The pupils did not respond to light, but were equal in size. A *tache cérébrale* was present.

On the evening of the following day, four days from the onset of the disease, he died.

The autopsy showed the convex surface of the entire brain and cord to be covered with a thick exudation of pus, the spleen to be enlarged, and the case to be one of acute cerebro-spinal meningitis.

A child, two years old, was brought to the hospital with the history that it had been showing symptoms of malaise for six weeks. Two weeks previous to entering the hospital it had a convulsion, and the indefinite and general symptoms had become more pronounced. There had been loss of appetite, with constipation, at times vomiting, slight cough, and a heightened temperature.

The head was retracted, and the muscles of the neck were rigid. The eyes were staring, but the pupils reacted to light. There was, at times, slight opisthotonos. The abdomen was retracted. The emaciation was extreme, so that the vertebræ and ribs became quite prominent. The child was apparently unconscious, and did not notice objects which were brought before its eyes, although the eyes were open. It moaned at times, and occasionally the legs were drawn up. No efflorescence was detected anywhere on the skin. The temperature varied from 36.6° to 38° C. (98° to 100.5° F.). Sometimes it cried out sharply, as though in pain. A *tache cérébrale* was often present.

The treatment of this case was simply the frequent administration of milk, with the addition of stimulants when indicated by the weakness of the pulse. The child was in so apathetic a condition that the use of any drug was unnecessary. Although at times it cried out as if in severe pain, yet these attacks were not sufficiently long to necessitate their control by an opiate.

During the following month the child remained in very much the same condition. The head was retracted at times, and the emaciation became extreme, the abdomen being very much sunken (boat-shaped). In the next two weeks the nourishment was taken more readily, the head was less retracted, and he began to notice objects around him, but he vomited once or twice nearly every day. The temperature at this stage of the disease became normal.

One month later, which was two months from the time when the child entered the hospital, he was able to sit up without help. There was no retraction of the head, but the muscles of the neck were very rigid, and the head showed a tendency to retraction.

During the following month the child continued to improve slowly, increased in weight, recovered his appetite, and when seen one month later was found on physical examination to be in a normal condition.

The following case of epidemic cerebro-spinal meningitis occurred in the service of Dr. Buckingham at the Children's Hospital.

The child was two years and eleven months old. She was breast-fed for fifteen months. At eight months she had scarlet fever ; at eighteen months she contracted measles. With these exceptions she had always been well and strong. Two days before entrance to the hospital she fell and struck her head. She complained subsequently of headache. The day before entrance she vomited several times, and held her hands to her right ear as if in pain. There was no history of disease of the ears, of retraction of the head, of rigidity of the neck, or of convulsions.

The physical examination showed nothing abnormal aside from a considerable degree of rigidity of the posterior cervical muscles and pain when passive movements of the head were made. There was some hypertrophy of the tonsils, with coated tongue and some secretion in the naso-pharynx. The temperature was 38.8° C. (102° F.), the pulse 130, and the respirations 32. A specimen of urine could not be obtained.

Two days later there was marked retraction of the head, with internal strabismus. The child was very irritable, and was evidently very tender on pressure over the back of the neck. On the day after entrance the ears were examined, and the auditory canals were found full of epithelial scales, which were removed, leaving the drum membrane and walls of the canal reddened. On the following day an acute serous effusion was noted in the middle ear. Paracentesis of the membrana tympani was performed and wicks inserted.

On the third day lumbar puncture was performed. The cerebro-spinal fluid was cloudy, with slight formation of fibrin. The cells were chiefly polynuclear leucocytes, with some mononuclear cells. Occasional organisms were found within the cells. Cultures from the fluid showed the presence of the diplococcus intracellularis. There were several single colonies, as well as fine, diffuse, pin-point growths between, on the culture-medium.

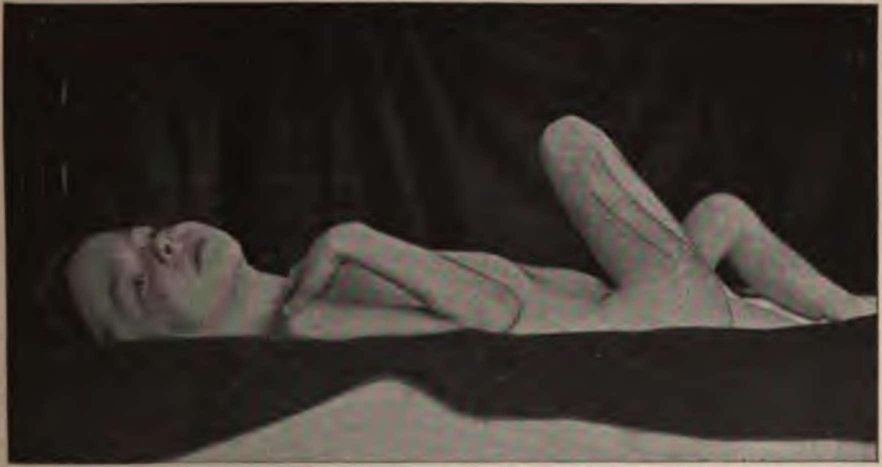
For six days the temperature gradually declined, and nearly reached the normal. The child sat up of its own accord, and freely moved its head. There was no strabismus. It then began to grow slowly worse. The temperature rose with considerable morning and evening variations, and for nearly a month ran a very irregular course, the evening temperature ranging between 39.4° C. (103° F.) and 40.4° C. (104.8° F.). The pulse varied from 120 to 146, and the respirations from 30 to 35. The aural symptoms disappeared, but the child grew more irritable ; retraction, tenderness, rigidity, and strabismus all returned. There was cough and an associated bronchitis. There were no convulsions or coma. The child was quiet but easily aroused. There was no general hyperæsthesia. The urine one month after entrance was examined and found normal. Loss of weight was very noticeable. Towards the end of the fourth week in the hospital the temperature began to fall, the symptoms gradually diminished, the mental and physical condition improved steadily, but there seemed to be a moderate degree of paralysis of the legs, which was slow in passing away. Convalescence was prolonged over a period of five weeks, when the patient was discharged to the convalescent home much relieved, but with the legs still not entirely under control.

The following case was that of a girl eight years old, who represented that form of cerebro-spinal meningitis which is designated chronic, and only a few cases of which have been reported.

Her parents were said to have been healthy, and there was no evidence of tuberculosis or syphilis in the family. A brother whom I saw in consultation died of cerebro-spinal meningitis. With the exception of an attack of measles and of whooping-cough, the child had not had any other diseases. She had not been entirely well since an attack of pertussis, which occurred one year previous to entering the hospital, and her attack of cerebro-spinal meningitis had begun four and a half months previously.

The onset of the attack was sudden. She went to bed in fairly good condition, but woke up in the night delirious, screaming, and apparently not recognizing her parents. These symptoms continued until the following week. There were no convulsions. A week later vomiting occurred every two or three days. This was not dependent upon food, and had occurred at intervals up to the time of entering the hospital. The bowels were constipated. There had been more or less opisthotonos from the beginning of the illness, and also in the beginning there was decided retraction of the head. The stiffness of the neck had gradually diminished, but at times returned. Up to the time of entering the hospital the child was said to have had constantly a heightened temperature, varying from 37.7° to 39.4° C. (100° to 103° F.), with a rapid pulse and quick respirations. Nothing abnormal had been found in the urine. There had been no efflorescence on the skin.

FIG. 108.

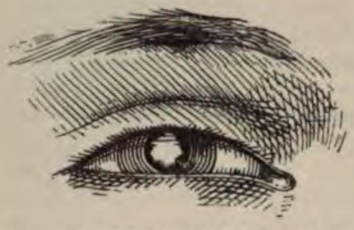


Chronic intermittent cerebro-spinal meningitis. Tache cérébrale showing on right thigh. Female, 8 years old.

Four weeks previous to entering the hospital the child was noticed to be blind. This had occurred suddenly. The child had had constant headache, and shortly after the beginning of the attack showed a loss of power of motion in both legs. At times there had been incontinence of fæces and of urine. An examination of the urine showed it to have a specific gravity of 1015, to be normal in color, to have an acid reaction, and not to contain albumin or sugar. No evidence of syphilis was detected. She sometimes showed improvement in her general symptoms and became conscious, but she was unable to sit up or to walk. As shown in Fig. 108, she was somewhat emaciated.

There was extreme hyperæsthesia of the body and extremities. The slightest motion of the bed caused discomfort and pain. An examination of the thoracic and abdominal organs showed that they were normal. The pulse was 80 and regular, the respirations were natural, the temperature was 37.7° C. (100° F.). She sometimes had an attack characterized by spasmodic contractions of all the muscles of the body, lasting for about thirty seconds. At these times there was no loss of consciousness, and the child screamed for some time afterwards as though in pain. During the attacks the pulse grew feeble and intermittent, the respirations slow and superficial, and the extremities cold. Brandy was given subcutaneously, and reaction took place. There was a decided *tache cérébrale*.

Fig. 109.



Secondary choroido-iritis occurring in cerebro-spinal meningitis.

Fig. 109 shows the condition of her eyes. The pupils reacted, and the retinæ were evidently sensitive to light, yet apparently she was blind.

In the middle of the eye was a yellowish mass with an irregular border. There was a very slight hyperæmia in the ciliary region. The iris seemed slightly pushed forward, and its pupillary edge was a little uneven. A yellowish or yellowish-white reflex appeared from the fundus of the eye even without the use of the ophthalmoscopic mirror, and it was not difficult to distinguish that this reflex did not come from the level of the lens, but that it was situated deeper. The tension of the eyeball was very much reduced, and there was very little tenderness on pressure.

These yellowish appearances in the pupils are sometimes called pus emboli, and also metastatic choroido-iritis; but while in other forms of meningitis metastasis may occur, it does not occur in the epidemic form, but is a direct extension.

In the other forms where it does occur, both the meningitis and the lesions of the eye are due to metastasis. Sometimes this yellowish mass fills the vitreous entirely, sometimes only in part. It may have blood-vessels on its surface.

The child remained in about the same condition for some time. At times she screamed as though in pain, but she took her nourishment fairly well. She had one slight convulsive attack, which involved mainly the upper extremities, the lower extremities being only slightly contracted. During this attack her thumbs were turned in, her fingers clinched over them, and her arms, which were usually extended at her sides, were flexed at the elbows. Her face showed no sign of spasm, and during the attack the radial pulse was full, soft, and regular. After a few seconds the muscles again became relaxed, and there was no further tendency to contraction. The usual position in which she lay during the following weeks was with the thighs slightly flexed and abducted, and the legs flexed at the knee, with the heels almost touching each other. About two weeks after entering the hospital the right leg became flexed on the thigh to such an extent that the knee almost touched the chin and the heel rested on the vulva. Any attempt to extend the leg made the child cry out as though in pain, the left leg being naturally extended in bed. This condition of the right leg continued for several days and then disappeared. Some days later a slight convulsive attack took place, which seemed to affect the right side more than the left.

The temperature during the eighteen days when the child was in the hospital varied from 37° to 38.8° C. (98° to 102° F.). The pulse during this time varied from 68 to 100; the respirations sometimes varied from 34 to 52, but were usually about 28.

The fingers were flexed most of the time, and there was so much rigidity of the limbs that the reflexes could not be satisfactorily determined. Later in the disease there was slight cyanosis of the cheeks and lips, and an eruption of milia on the chest apparently arising from her continually perspiring day and night. She lay in a stupor all the time, except when she was moved, when she would cry out. She showed no signs of understanding anything that was said to her. Sometimes she would be seized with an attack of rapid breathing lasting several hours. The bowels had been constipated at first, but later diarrhœa occurred. There was incontinence of fæces and urine, but no vomiting. The teeth were kept closed, and had to be forced apart when she was fed. Once she had a convulsion, in which the head was drawn back, the body and extremities were rigid, and the eyes rolled up.

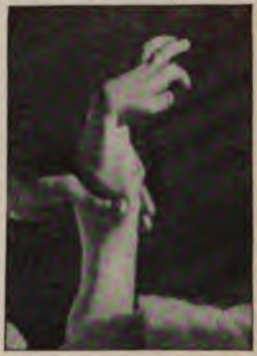
Eighteen days after entering the hospital the child was taken to her home, so that the daily record could not be obtained.

An examination made two weeks after she left the hospital showed a spastic condition of the extremities and neck.

When seen by Dr. Bullard at this time the child took no notice of her surroundings, and her eyes when opened had a vacant expression, due largely to the mental condition, although she was undoubtedly blind. The extremities were much wasted, and were all in a condition of spastic rigidity. There was slight flexion of the thighs on the body and of the legs on the thighs, while the feet were extended in nearly a straight line with the legs.

The hand, as shown in Fig. 110, was flexed almost at right angles to the wrist. The proximal phalanges of the fingers were hyperextended, while the other phalanges were flexed. The thumb was strongly adducted, and its distal phalanx was flexed.

FIG. 110.



Chronic cerebro-spinal meningitis. Spastic condition of hand five and a half months after onset of the disease.

This is a position of the hand frequently found in the later stages of spastic paralysis, and is due to the preponderant contraction of the flexors of the wrist and weakness of the interossei and lumbricales.

The child gradually grew weaker, and died of exhaustion a few weeks later.

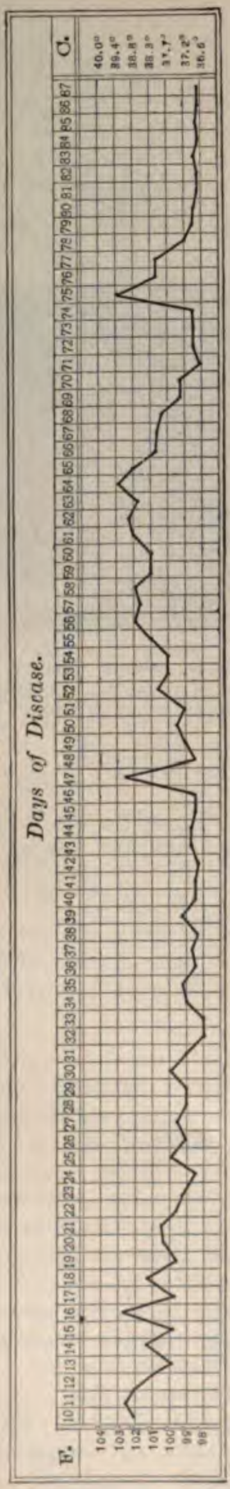
These chronic cases are rare. In two other cases which came under my observation the children eventually died from a prolonged sickness of many months, during which they at times seemed to be recovering. Cases have been reported by others, as by Henoch, of Berlin, to have recovered.

The following case was seen in consultation with Dr. Townsend:

A boy, four and a half years old, had been previously well, with the exception of an attack of measles when he was one year old.

On May 9 he was suddenly attacked with vomiting, which continued at intervals for two days. From the beginning of the attack he complained of severe pain in the head and abdomen. On the second day of the attack there was much contraction of the head, and he was slightly delirious, although rational most of the time. The temperature was raised from the beginning of the attack. There were no convulsions. The bowels were not moved during the first week of the disease. When first seen by Dr. Townsend the pulse was 124 and regular, the temperature 38.8° C. (102° F.), and the respirations 20 and regular. There was slight opisthotonos. There were no contractions of the muscles of the limbs. The symptom of Kernig was present. There was no tenderness along the spine. The cutaneous sensibility was everywhere normal. There were no cutaneous efflorescences or ecchymoses. The pupils were

CHART 11.



Chronic intermittent cerebro-spinal meningitis

regular and reacted normally to light. There was no strabismus or photophobia. Nothing abnormal was detected on physical examination. The child was apparently in great pain, cried out a great deal, and moaned continually. The suffering during the next few days was so great that morphine in doses of 0.002 gramme ($\frac{1}{30}$ grain) had to be given. This dose had to be increased so frequently that it was found that the child took 0.015 gramme ($\frac{1}{4}$ grain) before relief was obtained. Applications of ice to the head and spine gave no relief, and for a number of days it was found that there was needed to control the restlessness and pain 0.01 to 0.02 gramme ($\frac{1}{6}$ to $\frac{1}{3}$ grain) of morphine during the twenty-four hours.

On the twenty-fourth day of the disease the temperature, which had varied from 37.7° to 39.4° C. (100° to 103° F.), became normal, remaining so until the forty-seventh day. During this time the head was only slightly retracted, and the child seemed free from pain, but remained in a very listless condition, not speaking, and taking but little nourishment or stimulants. He became emaciated, passed his urine and fæces involuntarily, and occasionally vomited. Nutritive enemata were not retained, but on the forty-first day of the disease peptonized milk was retained, and on the forty-sixth day he was able to take gruel, and at that times talked and laughed.

On the forty-seventh day of the disease a relapse occurred, the temperature rising to 39.7 C. (103.6 F.). The head was rigidly drawn back, the eyes were staring, and the pain returned. The symptom of Kernig, which had never disappeared entirely, again became well marked. At this time I saw the child with Dr. Townsend. On the sixty-sixth day of the disease the convulsive movements of the left arm and leg, with turning in of the left eye, occurred. Several days previous to this relapse a number of sudamina appeared on the neck and trunk, and an evanescent erythematous eruption on the neck and face, lasting only a few hours. From the sixty-first to the sixty-sixth day of the disease his body was covered with a macular efflorescence, the macules varying in size. Ecchymoses were at no time seen, and repeated examinations of the chest and abdomen showed nothing abnormal.

From the seventy-first day to the seventy-third day the temperature was again normal, the child took his food well, the neck was straight, and his general appearance was encouraging.

On the seventy-fourth day he again had convulsive movements, most marked on the left side of the body. The head was drawn back, and at noon the next day his temperature was 39.4° C. (103° F.). The pulse, which during the entire illness ranged from 120 to 140 and had previously been regular, was now at times irregular and intermittent. The bowels were constipated at this time.

After this, although the temperature became normal, the child failed rapidly, and there was so much emaciation that the finger and thumb could easily encircle his thigh.

He died quietly on the eighty-seventh day from the time of the onset of the disease.

It was very difficult, indeed impossible, to give a prognosis in this case, as at times it seemed as though he would recover, and then the temperature would rise again and the unfavorable symptoms would return.

The chart shows the temperature from the tenth day of the disease.

TYPHOID FEVER.

Typhoid fever (*enteric fever*, *typhoidal ileo-colitis*) is an infectious disease produced by the bacillus of Eberth. The conditions and manifestations of typhoid fever as it appears in the fœtus, in the young child, and in the adult differ markedly.

Typhoid in the Fœtus.—The infection of the fœtus may take place from the mother. Abortion occurs in from fifty to seventy per cent. It is now well established that the bacillus of typhoid can pass through the placenta from the mother to the fœtus, and it seems probable that this can happen when the placenta is intact. In most cases the fœtus dies in utero; it may, however, be born alive but suffering from the infection. Again, the fœtus may be born alive and healthy, after having passed through the infection in utero. Finally, it may not be infected at all, although the mother may have had the disease. When at birth the fœtus is infected it rapidly succumbs, the infection showing itself in the form of acute cachexia without any special characteristics. The bacilli in these cases are most commonly found in the liver, spleen, and blood; they may also be found in the intestine. Marked lesions in the intestine and mesenteric glands are, however, never found, the organs usually being normal. The liver and spleen are sometimes swollen, and may show minute hemorrhages. An explanation of these lesions is found in the entrance of the bacillus through the umbilical veins,—that is, the septicæmia is primary and not secondary to an enteritis. These cases correspond to those of typhoid fever in adults in which there are no intestinal lesions. There may also be an infection with other organisms besides the typhoid bacillus, especially the staphylococcus aureus.

Typhoid in Infancy and Early Childhood.—The pronounced pathological lesions, severe symptoms, and great virulence in type which are so characteristic of typhoid fever of later years are so rare in infancy and early life that the following description will be confined to the latter period.

ETIOLOGY.—Typhoid fever is exceedingly rare in the first two years of life, is uncommon under three years, and after the fifth year becomes more common as the child grows older. Under two years the number of cases reported, even if correct, is small. In fifty-five cases collated in 1895 by Stowells, the diagnosis in twenty-eight seemed probable; in the others it was doubtful, as no bacteriological examination was made. The youngest authentic case of typhoid contracted after birth is that reported by Gerhardt in an infant which presented the symptoms when twenty-five days old, and recovered in twelve days. In Stowells's series sixteen confirmative cases have been reported. Eight of these cases died. Infection may take place in any way by which the germs of the disease can be carried from one individual to another. The germs are given off from the discharges, chiefly the fæces and the urine. The individual is infected largely through the gastro-enteric tract, and the most usual mode of conveyance into the body is by infected milk or water. Direct transmission by the hands which have been infected from the surroundings of the patient, especially from the clothes and bedlinen, is possible.

Pathology.—After birth in the earlier periods of life the pathological lesions are less definite in direct ratio to the age. After the second or third years the lesions are constant and the pathology definite. Although the more advanced and severe lesions of typhoid fever may occur in the early as well as in the later years of life, yet its characteristic lesions in young subjects are found in the milder and less severe pathological changes of the disease. These consist essentially of a hyperplasia of the solitary lymph-follicles and Peyer's patches, and the process, instead of going on to ulceration, usually terminates in early resolution with fatty degeneration of the cells. Hemorrhage and perforation are therefore rare complications in the typhoid fever of early life. There is, however, nothing distinctive of typhoid fever in this hyperplasia of the lymph-follicles in children, for it is not uncommon to find this condition when death has occurred from other diseases of the intestine. It may also be present in such diseases as measles, diphtheria, and scarlet fever. Very marked hyperplasia of the lymph-follicles may be produced in children by irritating substances and by foreign bodies, not only food, but also drugs, such as turpeth mineral. I have, in fact, seen, at the post-mortem examination of a child, marked enlargement of the lymph-follicles caused by doses of turpeth mineral given during life as an emetic.

In addition to the lesions in the intestine, hyperplasia of the mesenteric glands, granular degeneration of the liver and kidney, and granular or fatty degeneration of the heart may be found. Parenchymatous changes sometimes occur in the peripheral nerves and degeneration of the muscles. The pathological conditions in typhoid fever in the early years of life may be said to correspond with those which are met with in the aborted forms of the disease in later life.

The specific efflorescence of typhoid fever, the rose-spots, have acquired additional importance in the diagnosis from the recent bacteriological investigations by Neufeld and Curschman in Germany, and Richardson in this country. These observers have demonstrated the presence of the typhoid bacillus in the rose-spots in a very large proportion of the cases examined. In Richardson's series the diagnosis was made by this method on an average of six days before the appearance of the Widal reaction.

INCUBATION.—The stage of incubation in typhoid fever may last from one to two weeks; a shorter time, however, five days, and a longer one, three weeks, have been observed.

SYMPTOMS.—The prodromal stage is usually short, young subjects having less power of resistance to the poison than adults, in whom the prodromal stage is often prolonged. The onset is more likely to be acute in young children, and nervous symptoms, especially vomiting, are common.

The duration of the disease is generally much shorter than in adults. This short duration depends largely upon the mild form of the intestinal lesions and usually indicates that marked ulceration has not taken place. As shown by the figures collected by Morse, the average duration of typhoid fever in children is a little less than three weeks, the younger the child the shorter the duration, and the proportion of cases in which the duration is ten days or less is twice as great in the younger children.

As a rule, the temperature is moderate, but it may be high, as in adults, without, however, producing as severe symptoms, since children are generally less affected than adults by a high temperature. When the lesions are mild the temperature is not apt to show so typical a course as when they are pronounced. The temperature often rises rapidly in the beginning, and although it returns to the normal by lysis, it does not show so gradual a lysis as when marked lesions have occurred, and when other symptoms of toxæmia have complicated the disease. The younger the child the lower the range of the temperature and the sooner is the maximum reached. The remittent stage occurring in adults is much less in children and the defervescence is rapid.

The pulse is usually accelerated, but is low in comparison with the height of the temperature in other diseases, and is not so apt to vary from the normal in strength and rapidity. Hæmic murmurs are not uncommon.

The respirations are not especially increased unless some complication is present. Although bronchial catarrh may in most cases be pathologically present (Morse), yet clinically its importance in children has been rather over-estimated. In certain cases in young children, however, it is so marked as to be liable to lead to the error of considering the primary cause of pulmonary origin rather than intestinal.

The abdomen may be distended and tympanitic, but this symptom is often not marked. In Morse's statistics distention occurred in from 50 to 70 per cent. of all cases. It is more common in older children, and not infrequently is extreme. Pain and tenderness of the abdomen are rather unusual. Vomiting, especially at the onset of the disease, is often met with, but is rare in the later stages of the disease. Constipation, especially in young children is more common than diarrhœa. The discharges are not so characteristic as in adults, and when diarrhœa is present, the stools, as a rule, are not so frequent or so large in amount. All the fatal cases noted by Morse had diarrhœa. Intestinal hemorrhage is very unusual under ten years of age, and is fatal in about half the cases. Perforation is exceedingly rare. Although it is probable that in all cases of typhoid fever there is some enlargement of the spleen, it is often impossible to detect this change by palpation, and percussion of the spleen in young children is well known to be very misleading. According to Morse's figures the spleen is demonstrably enlarged in 90 per cent. of all cases of typhoid in children, and the older the child the greater the enlargement. The return of the spleen to its normal size indicates convalescence and recovery, while persistent enlargement shows that the disease is still present.

Epistaxis occurs more rarely than in adults.

The rose-colored spots appear to be less frequently found in children than in adults. Morse's series of cases showed the spots in from 60 to 70 per cent. of all cases in children. They usually appear in the second week. They consist of small, light-pink maculæ, usually scattered, chiefly or solely on the abdomen, and disappearing under gentle pressure of the finger. Other varieties of efflorescences are seen at times, but are uncommon. Herpes labialis occurs in some cases.

The tongue is not so likely to be dry as in older subjects, and although coated it soon becomes clean at the tip and edges. There is at times a slight albuminuria at the height of the fever, but a complicating nephritis is rare. The diazo-reaction is usually present in the urine after the first week. The Widal reaction can generally be obtained in the second week.

The more severe nervous symptoms so pronounced in later life are not prominent in early childhood. Headache of a mild type is common; severe headache is very uncommon. Crying out at night, especially in young children, and delirium of a mild type are not uncommon. Deep stupor is rare; in fact, the characteristic of typhoid fever in young children, as I have seen it, is apathy. The child takes the nourishment which is given to it, is not especially restless, and usually lies in a half-somnolent condition. As the disease progresses it gradually returns to a more natural mental condition. Convulsions are rare. In some cases cerebral symptoms, simulating somewhat those of meningitis, arise, and are frequently due to cerebral congestion or to toxic action. These symptoms are more common in younger children. Hyperæsthesia, changes in the pupils, retraction of the head, and opisthotonos may be present, closely simulating meningitis. True meningitis caused by the bacillus of typhoid is rare, but can occur. A case of this kind with the bibliography has been reported by Wentworth in a girl four years of age, in the Transactions of the American Pediatric Society of 1899. Lumbar puncture in this case showed an enormous number of typhoid bacilli, but no other organisms were found. The higher the temperature the more pronounced are the nervous symptoms.

In typhoid the leucocytes are usually diminished in number, but there is a proportionate increase in the lymphocytes. In the early stages the erythrocytes are increased, as is also the hæmoglobin. In the later stages a condition of secondary anæmia may occur, producing a diminution of the red blood-corpuscles and hæmoglobin.

COMPLICATIONS AND SEQUELÆ.—Pulmonary complications, with the exception of bronchitis, are rare. Neuritis is not an uncommon complication. Aphasia occurs rather more frequently in children than in adults. In rare cases it may be due to some organic cause, such as embolism, but, as a rule, there is no demonstrable lesion. According to Morse's statistics, in most cases it occurs where the typhoid had been extreme, accompanied by other marked nervous manifestations, and coming on during convalescence while the temperature was falling. The intelligence is retained, and there is rarely any other paralysis. Recovery was the rule, and the average duration was three weeks. Even when partial paralysis has accompanied the aphasia, entire recovery may take place. Chorea and parotitis are not very uncommon, and otitis media is not infrequent. Relapses are about as frequent as in adults. According to Morse's figures, the number of days of fever in thirty relapses was fifteen, the shortest seven days, and the longest twenty-nine days. His number of days of apyrexia before the relapse varied from none to nineteen days. They follow about the same course as the normal attack, but, as a rule, are of shorter duration, the symptoms being less severe.

DIAGNOSIS.—*Widal Reaction.*—During the first few days typhoid fever may often be mistaken for various forms of febrile gastro-enteric disease. The most important means of diagnosis is by the Widal serum-reaction. By this is meant the clumping and loss of motility of the typhoid bacilli when the serum of a patient sick with typhoid fever is added to a bouillon culture of typhoid bacilli. A freshly prepared bouillon culture not more than twenty-four hours old and grown at room temperature should be used. Not less than ten parts of the culture should be added to one of the serum. No reaction occurring after half an hour should be accepted. This reaction is usually not present until the beginning of the second week, but may last for weeks or months after the recovery. If, therefore, a reaction is positive, it is necessary, in order to be accurate, to exclude a recent typhoid. While this is the most important method of diagnosis in typhoid fever, it is not absolute, as the reaction is not always present in typhoid, and may rarely occur in other diseases. The error is about five per cent. in either direction.

Infants born alive or dead of mothers who have typhoid may or may not show the serum-reaction. The reaction may be transmitted to a healthy infant from the breast-milk. The reaction is less intense in the mother's milk than in her blood, and less in the infant's blood than in the milk. The agglutinating principle may not be transmitted to the infant through the milk. The transmission probably depends largely on the strength of the agglutinating power in the blood and milk. The reaction disappears in a few days after the cessation of nursing.

Diazo-Reaction.—Not so much value as formerly is attributed to the diazo-reaction, because it is also found in acute tuberculosis, septicæmia, measles, pneumonia, and probably also in many of the acute febrile diseases. It does not develop, however, in acute tuberculosis until after the third week of the disease; whereas in typhoid, if it is to appear at all, it is usually obtained by the end of the first or in the second week.

White Blood Count.—Another important aid in the differential diagnosis of typhoid may be obtained by the white blood count. The *absence of leucocytosis* is the characteristic point of the blood in typhoid, and serves to exclude many other diseases in which leucocytosis is present. It is always to be borne in mind, however, that a high white blood count may be found in a case of typhoid which is complicated by some disease which is accompanied by a leucocytosis. Typhoid fever, therefore, is not to be excluded because of a leucocytosis, unless complicating conditions can positively be proved to be absent.

Typhoid Bacilli in the Rose-Spots.—The detection of the typhoid bacilli in the rose-spots is a point in the differential diagnosis which may be attempted in all doubtful cases; their presence makes the diagnosis certain. In making the differential diagnosis, therefore, on a doubtful case of typhoid, the most important evidence is to be found in the results of the Widal and diazo-reactions, the white blood count, and the rose-spots. The onset of the disease, the course of the temperature, and the whole series of abdominal symptoms must be considered as supplementary and confirmatory evidence.

The diseases which are most likely to simulate typhoid fever and render the diagnosis difficult are as follows:

The Exanthemata and Pneumonia.—A number of acute diseases, such as the exanthemata and pneumonia may simulate in their prodromal stage those cases of typhoid fever which begin with violent symptoms. In pneumonia and scarlet fever there is leucocytosis, in typhoid fever there is none; moreover, in the former diseases there is no Widal reaction and no diazo. In measles, however, there is no leucocytosis, while the diazo-reaction may be present. After the prodromal symptoms have passed, the distinctive efflorescences of measles and scarlet fever and the course of the disease will serve to differentiate them, while the temperature curve and physical signs will determine the presence of pneumonia.

Acute Ileo-Colitis.—Typhoid fever and ileo-colitis may sometimes simulate each other. The intestinal symptoms of typhoid fever in young children, however, correspond so closely to the severity of the local lesions that it is rare in the early days of the disease to meet with the severe intestinal symptoms so common in ileo-colitis; those of typhoid, as a rule, being much milder. Later in the disease the splenic enlargement, efflorescence, and tympanites of typhoid are not found in ileo-colitis. The blood of typhoid does not show leucocytosis, while this is usually present in other forms of gastro-enteric infection. While the Widal and diazo-reactions are generally present in typhoid, the former never occurs, and the latter rarely, in gastro-enteric disease. The temperature curve also, after the early days of the disease, is of considerable aid in diagnosis.

Tubercular and Epidemic Cerebro-Spinal Meningitis.—The vomiting which occurs in the prodromal stage of typhoid fever may, in connection with the child's apathy, simulate the early stages of tubercular meningitis, but the higher temperature, enlarged spleen, and distended abdomen of typhoid, and the absence of the irregular respirations, slow, intermittent pulse, and retracted abdomen of meningitis serve to differentiate the two diseases. In tubercular meningitis the evidence given by lumbar puncture and the absence of the Widal reaction separate the disease from typhoid. In both diseases the diazo-reaction may be present, and leucocytosis is absent in both. In rare cases the bacilli of typhoid, as already stated, may be the cause of a meningitis and the disease be diagnosticated during life by lumbar puncture. In cerebro-spinal meningitis the diplococcus intracellularis, obtained by lumbar puncture, is not found in typhoid; there is marked leucocytosis, and while the Widal reaction is always absent, the diazo may be present.

Acute Miliary Tuberculosis.—The most difficult of all the diseases to differentiate from typhoid is acute miliary tuberculosis. The absence of leucocytosis and the possible presence of the diazo-reaction occurs in both diseases, but the Widal reaction is not present in tuberculosis, and thus becomes invaluable in diagnosis. Unless the tuberculin reaction is marked, and until other evidence of tubercular processes appear locally, as in the lungs, it is often only by the subsidence of the fever and prostration in typhoid at an early date that it can be distinguished from the slow progressive prostration and continued fever of tuberculosis. It is especially in the typhoidal type of acute miliary tuberculosis that the resemblance of the two diseases is closest, and when in typhoid the rose-colored spots do not appear and the Widal reaction is negative, the delirium, distended abdomen, enlarged spleen, and even the irregular temperature at times common to both diseases, may make the resemblance so close that the diagnosis cannot be made until the post-mortem examination.

Malaria.—The differential diagnosis between typhoid fever and malaria is, at times, especially in children under two years of age, very difficult and perhaps impossible until the blood has been examined. The difference in the temperature curve, the presence of the plasmodium, the marked effect on the symptoms by the use of quinine, and the absence of the Widal and diazo-reactions in malaria, are of much value in the diagnosis from typhoid. Leucocytosis is absent in both malaria and typhoid.

Epidemic Influenza.—Epidemic influenza may in its onset simulate typhoid fever, but the period of doubt is very short. The presence of Pfeiffer's bacillus, if it can be found in the sputum or nasal discharge, is of importance, but the absence of the Widal and diazo-reactions are not of so much importance, as the disease has either run its course or declared itself by its temperature and irregular groups of symptoms before the time for obtaining these reactions has arrived. It is, therefore, impossible in the early days of the disease to make a positive diagnosis, and in some cases we are left in doubt as to the diagnosis at even a later period, when the symptoms of enlarged spleen, rose-colored spots, and tympanitic abdomen of typhoid are not present as a group. The white blood count is of little service, as it is generally normal in epidemic influenza.

Prognosis.—The prognosis of typhoid fever in early childhood is good. Statistics show that the disease is much less fatal in children than in adults, and that the mortality increases directly with the age. The complications, whether arising from local disturbance of the intestine or from cardiac and pulmonary disease, are rare in comparison with those met with in later life. The disease, however, varies very much in its severity in different epidemics and in different stages, and a child may have a severe type of typhoid fever and die from it.

Treatment.—The treatment of the mild form and that most commonly met with in young children is exceedingly simple. The child should be kept perfectly quiet in bed as long as the temperature is raised, and for three or four days after it has become normal. Although in adults quite a varied diet can be given, in young children milk in some form throughout the whole course of the disease is indicated. There are no drugs which will either abort or cure the disease. In the cases which begin acutely with vomiting, food should be withheld for some hours, and the prodromal symptoms treated symptomatically, as the same symptoms would be in the beginning of a number of acute febrile diseases. After the acute symptoms have passed, alkaline milk should be given with low percentages of fat, sugar, and proteids at first, and these elements should be gradually increased in their percentages, and varied in their combinations to suit the especial age and digestion. It is usually better to heat the milk to 68.3° C. (155° F.) and to add lime-water, five per cent. Where the stomach is at all irritable it is often well to peptonize the milk for a time. It is seldom that the temperature is so high as to call for anything beyond bathing, and, as a rule, antipyretics should be avoided. As a hygienic measure the child should be bathed twice daily with water heated to 32° C. (90° F.). If the temperature rises to 39.4° or 40° C. (103° or 104° F.) a reduction by artificial means is not necessarily indicated, but we should be guided by the general condition of the child. According to our experience at the Children's Hospital, a large number of children of all ages pass through an attack of typhoid without any necessity arising for the reduction of the temperature by baths or sponging. The older the child the more likely is the bath to be indicated. Much pulmonary or cardiac weakness contra-indicates the bath. The child can be either sponged for five or ten minutes, or wrapped in a thin blanket and placed in the bath, while the body and limbs are actively rubbed. Where there is delirium and great restlessness, with a temperature of 40.5° or 41.1° C. (105° or 106° F.) baths should be given every three or four hours, but with caution, as children do not react so well as adults. A stimulant, such as a few drops of brandy, should be given at the time of the sponging or bath.

Technique of the Bath in Typhoid Fever.—A very convenient method of administering the bath in typhoid fever, or in any other condition in which the treatment is indicated, is as follows:

A large rubber sheet is placed under the child on the bed, and the sides and ends are bolstered up by rollers or bedding, making, if properly arranged, a shallow but sufficiently large tub without the inconvenience of moving the child. A towel or blanket may be placed under the child if it is made uncomfortable by lying directly on the rubber sheet. The water is brought in a foot-tub to the side of the bed, and its temperature regulated according to the indications. A wet towel or sponge is placed on the head; the water is then squeezed from a sponge on to the naked body with one hand, and brisk friction is applied with the other. The chest, abdomen, arms, and legs should be taken in rapid succession. It is especially to be noted that it is the combination of *friction* with water which accomplishes the best result in reducing the temperature and at the same time stimulating the peripheral circulation. The child is then turned on its side, and the back is bathed and rubbed in the same manner. The duration of the bath should vary from five to fifteen minutes, according to the height of the temperature and the manner in which the child reacts. The temperature of the water should vary from 37.7° C. (100° F.) to 23.8° C. (75° F.), according to the age of the child, the height of the fever, the number of degrees of reduction in the temperature obtained, and the manner in which the child reacts. A drop in the temperature of one to one and a half degrees Centigrade is the object especially to be attained. The temperature is best taken one-half hour after the bath. Drops of two and three degrees are not desirable. If the bath is well given by an experienced nurse, a good reaction should usually be obtained. Slight blueness of the lips and finger-nails, cold hands and feet, and chilly sensations should be watched for and treated by a dose of brandy and water, an extra blanket, and heaters in the bed. Massage of the extremities under the blankets may be all that is necessary.

Care of the Mouth and Skin.—The mouth should be gently but thoroughly cleansed with a two per cent. solution of boracic acid in distilled water at least three times daily. The greatest care should be taken to prevent irritation of the skin by bathing the recumbent parts with alcohol and water.

Diarrhœa, unless profuse and exhausting or very frequent, such as more than three or four in twenty-four hours, should not be checked. To control peristalsis bismuth and small doses of opium are indicated.

Constipation.—There is usually more difficulty with constipation than with diarrhœa. Enemata and suppositories are indicated rather than laxatives, which should be only used in extreme cases, and necessarily sparingly and in a mild form, such as the milk of magnesia 3.75 to 6.50 c.c. (1 to 2 drachms), with an occasional small dose of castor oil. The discharges should be disinfected at once with a 1 to 20 solution of carbolic acid. All the clothing should be soaked in carbolic acid, twenty per cent. for six hours, and then boiled for one hour.

Headache, Restlessness, Sleeplessness.—These nervous symptoms are best treated by bathing. If a sedative is desired, 0.12 to 0.3 gramme (2 to 5 grains) doses of trional are safe and efficient. Bromide of soda may be given alone or in combination with trional.

Tympanites should be treated with hot cloths applied to the abdomen, or by turpentine stupes made in the following manner: one part of spirits of turpentine is mixed with six parts of sweet oil, and the mixture is then rubbed gently but thoroughly on the abdomen. A thick piece of flannel is then dipped in hot water, wrung out, and when sufficiently cool applied to the abdomen, covered with oiled paper, and kept in place by a thick swathe. If these precautions are taken, irritation of the skin will rarely be caused, and the treatment is very efficacious. Gentle irrigation of the colon with some saline solution, such as a two per cent. borate of soda or a three per cent. boracic acid solution, may be tried. In extreme cases the rectal tube may be used with caution.

Hemorrhages and *perforations* should be treated as in the older cases, the former with absolute rest, with hypodermic injections of ergotin 0.06 c.c. (1 grain). and sometimes of morphine and cold to the abdomen, and the latter with hypodermic injections of morphine 0.001 to 0.003 c.c. ($\frac{1}{60}$ to $\frac{1}{20}$ grain).

Laparotomy for perforation in typhoid has not been attended by brilliant results, but in certain cases may be deemed advisable. The same care should be exercised during the *convalescence* of the child as in the advanced convalescence of the adult.

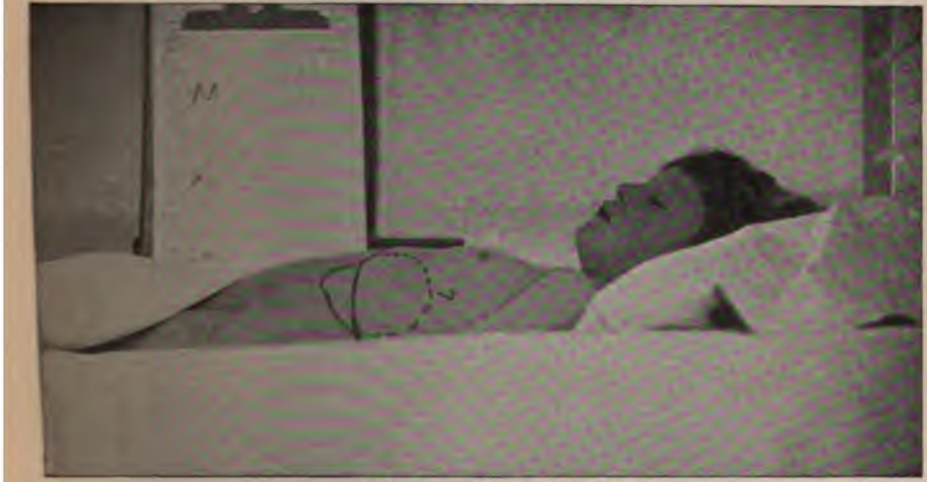
Stimulants are not called for, as a rule, in the early stages of the disease. When used they are best administered in the form of brandy or whiskey. They are contra-indicated in the stage of toxæmia represented by marked excitement, active delirium, cerebral congestion with delusions and great restlessness, and a strong, full pulse. On the other hand, when the pulse is small and weak, when the mental condition is expressed by depression, low muttering, delirium, and a general condition of nervous exhaustion, the use of stimulants, especially in the form of alcohol, is distinctly indicated, and is highly beneficial. For an infant of one year the amount of brandy or whiskey in twenty-four hours should vary from 15 c.c. ($\frac{1}{2}$ ounce) to 30 c.c. (1 ounce), but should never exceed 60 c.c. (2 ounces). In children of four years these doses may be doubled in corresponding conditions. In all cases the stimulant should be well diluted.

If in a hemorrhage from the bowels a stimulant must be given, it should be administered in small doses and in combination with opium. A table giving the doses of other cardiac stimulants will be found on page 470.

The following case, Fig. 111, was under my care at the City Hospital:

A boy, five years old, was taken sick with general malaise and fever five days before entering the hospital. There had been no other symptoms, such as epistaxis or vomiting. On entering the hospital, an examination showed the tongue to have a thick brownish coat in the centre and a thin coat on the tip and edges. The child was in an apathetic condition. The pulse was rapid and regular. Nothing abnormal was found in the thorax. The abdomen was distended and tympanitic, and showed one rose-colored spot. The spleen could be easily felt 2.5 cm. (1 inch) below the border of the ribs, and on percussion the dulness reached as high as the seventh rib in the axillary line. The enlargement of the spleen and the lower border of the ribs are marked in black in Fig. 111. The upper border of the splenic dulness is marked by a broken line, and the figure 7 marks the seventh rib. The blood showed no leucocytosis. The pupils reacted equally to light. The expression of the child's face was apathetic, and he took very little notice of anything. An examination of the urine showed the color to be normal, the reaction neutral, the specific gravity 1026, and that there was a slight trace of albumin. The sediment showed occasional hyaline and fine granular and fibrinous casts. A Widal reaction was not obtained.

FIG. 111.

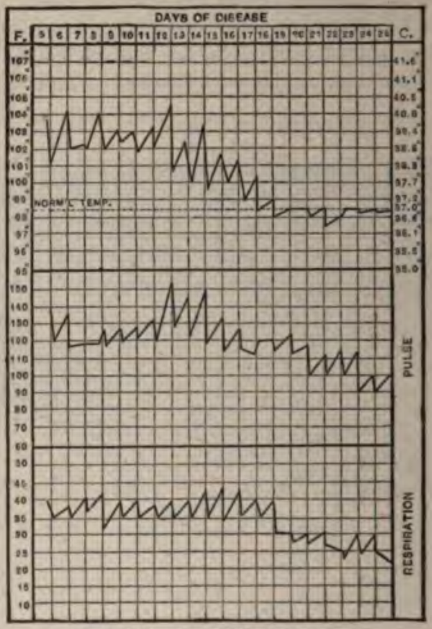


Typhoid fever. Male, 5 years old.

On the third day after entering the hospital, the eighth day of the disease, the child became very stupid and sometimes delirious. There was a slight cough. On the twelfth day of the disease the child cried out at times, and was delirious. The skin was dry and hot. There were no more rose-colored spots. There seemed to be slight tenderness in the lower iliac fossa, but there was no gurgling. On the fifteenth day of the disease the temperature began to fall by lysis, and the child began to be fretful. On the eighteenth day the temperature became normal. By the twenty-first day the child seemed bright, and was playing with its toys. The pulse was stronger. One week later it was sitting up in bed, and had a strong pulse and a good appetite. A few days afterwards it was up and about the ward, perfectly well. The following chart shows the temperature, pulse, and respiration from the fifth day of the child's illness until convalescence was established on the twenty-fifth day.

The following notes and plate (Fig. 112) were given to me by Dr. S. S. Adams, of Washington, and occurred in an infant two years old. In this case the irregularity of the temperature curve and the prominent symptoms of cerebro-spinal irritation rendered the diagnosis so obscure that typhoid fever was not suspected until a few days before death. The post-mortem examination showed marked congestion of the entire brain, chiefly on the right side. The left hemisphere was covered with a glue-like substance which filled the sulci and was especially abundant around the Sylvian fissure. The heart was normal. The lungs showed marked hypostatic congestion. The liver was normal. The gall-bladder was empty and pale. The spleen was enlarged. The kidneys were normal. The stomach was congested. The mesenteric glands were enlarged and soft. The intestines contained a quantity of yellowish watery fæces. The lesions were in the ileo-colic portion of the intestine. There was thickening and ulceration of Peyer's patches, and to a less extent of the solitary follicles.

CHART 12.

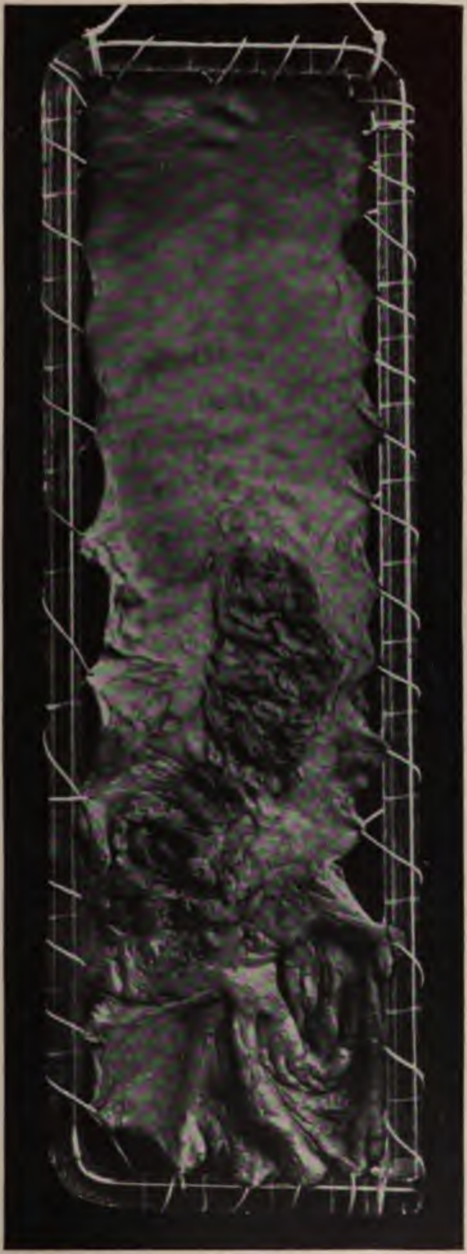


Typhoid fever. Male, 5 years old.

Swelling of Peyer's patches and of the solitary follicles is not distinctive of typhoid fever, as this condition frequently occurs from irritations of various kinds. Fig. 113 represents a section taken from the intestine of a child.

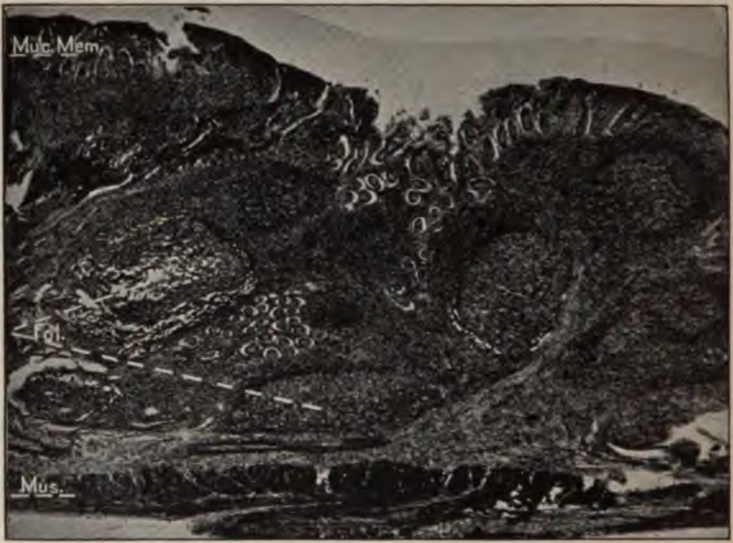
The macroscopic appearances of this intestine so closely simulated the early stage of typhoid fever that cultures were made from it by Dr. Prudden to settle this question. No bacilli were found. This condition is often found in children in acute non-typhoidal ileo-colitis.

Fig. 112.



Typhoidal ileo-colitis, showing ulcers of colon. Female, 2 years old.
U. S. Army Medical Museum.

FIG. 113.



Enlarged Peyer's patches closely simulating the lesions of typhoidal ileo-colitis. Muc. Mem., mucous membrane; Fol., enlarged follicles; Mus., muscle.

TYPHUS FEVER.

Typhus fever is an acute infectious disease, the cause of which has not yet been determined. In America typhus fever is an exceedingly rare disease, but in England it is well known to occur in children as well as in adults. According to Collie, out of 711 admissions to the Hunnerton Fever Hospital during a period from 1871 to 1880, 24 were under five years of age, 54 from five to nine years, and 113 were from ten to fourteen years. Buchanan has shown that typhus fever in early life is very mild, but that children are just as susceptible as adults.

Symptoms.—The onset is acute. According to Ashby and Wright the attack begins with headache, pains in the limbs, drowsiness, sometimes vomiting, and rarely diarrhœa. The constitutional symptoms are of a severe type. The tongue is dry and coated, there are sordes on the teeth, and the gums bleed easily. On the fourth or fifth day the skin assumes a dusky, congested appearance, with an indistinct mottling. Sometimes there are rose-colored spots or maculæ, larger than those seen in typhoid, appearing first, according to Collie, on the upper front portions of the chest, on the wrists, on the backs of the hands, and in the epigastrium.

The temperature is usually continuously high, varying from 39.4° to 40° C. (103° to 104° F.), and the pulse 120 to 130, and rather weak. The temperature usually becomes normal by about the eighth or tenth day.

Prognosis.—The prognosis is usually good. Out of 191 children under fourteen years of age only two deaths took place, while the total mortality for all ages was 20 per cent.

Treatment.—Especial attention should be paid to the hygienic surroundings, such as fresh air, sunlight, and warmth. Daily sponging and the free use of stimulants are indicated. The diet should be milk and broths until convalescence is well established.

DIPHTHERIA.

Diphtheria is an acute, highly infectious disease, due to the Klebs-Loeffler bacillus. It is primarily a local affection, the constitutional symptoms being due not to the presence of the organism in the blood, but to the absorption of the toxins caused by the growth of the bacillus. Although the term diphtheria, derived from the Greek word *διφθέρα*, implies a membrane, and the disease is usually accompanied by a pseudomembrane, yet it must be understood that the disease is not diphtheria unless the specific germ is present. This germ may act with different degrees of intensity, and thus cause inflammation varying from a slight catarrhal condition to the most serious membranous one. In like manner, according as the form is mild or severe, there may be very slight constitutional symptoms or exceedingly serious ones. There is probably a concurrent infection with pyogenic cocci. These organisms produce the secondary inflammations occurring in the disease, and also by their toxins give rise to additional constitutional symptoms.

ETIOLOGY.—The Klebs-Loeffler bacillus was first described by Klebs in 1883, and later was more fully identified by Loeffler in 1884. Its most striking features, morphologically, are its variation in form and the irregularity in its manner of staining. It does not form spores. Welch and Abbott have shown that in fluids it may be killed by an exposure of ten minutes to a temperature of 58° C. (136.4° F.). Under favorable conditions it may remain alive for weeks, or even months, in fragments of dried membrane.

The pathogenic cocci most frequently found in the concurrent infections are the streptococcus pyogenes alone, or associated with the staphylococcus pyogenes aureus, the former being the most important in its action. There is no true diphtheria where the Klebs-Loeffler bacillus is not present, but its presence in a healthy throat does not constitute the disease diphtheria, although the individual may be the source of infection to others. It is undoubtedly a contagious disease of local origin, and it does not originate spontaneously. The contagium of diphtheria is contained chiefly in the secretions of the throat and nose, and is communicated usually by direct or indirect contact, and not, as a rule, by the air. The area of infection is not so great as in scarlet fever. It is not found in the urine and fæces. Sewer gas and confined impure matter of any kind may act by weakening the resistance of the body to the action of the bacilli, or, by producing a benign lesion in the throat, may offer a suitable nidus for the invasion of the bacillus. It is now, however, conceded that imperfect drainage and unsanitary conditions should not be considered important factors in increasing the frequency of the disease. Animals, such as cows, cats, dogs, and pigeons, may have diphtheria, and be a source of infection to those brought in contact with them. There is no positive evidence that the milk of cows affected by diphtheria contains the germs of the disease, but the probability is that the milk in some of these cases may be contaminated by the hands of the milkers. The cases in which the pharynx and nose are affected are the most contagious on account of the secretions. Generally an unhealthy condition of the mouth, nose, or throat predisposes to the disease, as a lesion of the mucous membrane is necessary for its entrance. The bacilli will not grow on an intact mucous membrane. An abrasion must be present for them to develop.

There is a non-virulent form of the diphtheria bacillus which differs in a number of respects from the virulent form, both in its culture peculiarities and in its method of staining with Hunt's differential stain, and also with Neisser's stain.

Although diphtheria may occur at any age, it is rarely met with in early infancy. It is most commonly seen from the second to the fifth or sixth year. It may occur more than once in the same individual.

Pathology.—The lesions of diphtheria are due to the local action of the Klebs-Loeffler bacillus and associated bacteria, and to the absorption of toxins produced by the bacteria. The most constant lesion is a pseudo-membrane, produced by the local action of the bacilli in the upper air-passages, and due to exudation and coagulation necrosis. The exudation coming from the vessels of the tissue beneath the membrane meets the necrotic tissue containing a fibrin ferment, and coagulation takes place, the fibrin of the coagulum being intimately associated with the necrotic epithelium. The bacilli can also produce other lesions, such as simple inflammation, necrosis without membrane formation, and even abscess. There is no pathological condition directly characteristic of the action of the Klebs-Loeffler bacillus, and the same anatomical conditions may be caused by other bacteria, and even by irritants. The process may be simply a catarrhal inflammation, which does not go on to the formation of a pseudo-membrane.

The primary infection in diphtheria is most commonly in the throat, from which the bacilli may extend into the adjacent mucous surfaces, producing, in some cases, membrane formation, in others only simple or purulent inflammation. They may also extend into the nose, producing nasal diphtheria, or along the Eustachian tubes into the middle ear, or from the nose into the accessory sinuses, downward into the trachea and air-passages, or into the œsophagus and stomach. There are certain differences in the membrane formation due to the anatomical character of the tissue. Thus the membrane in the pharynx clings tightly to the surface, while in the trachea and lower passages it is easily removed.

The extension of the bacilli into the lungs may produce a membrane formation in the smaller bronchi, areas of broncho-pneumonia or even abscesses. The pathological process may be further modified by the association with the Klebs-Loeffler bacillus of the common pathogenic cocci, and it is difficult to determine exactly the part which these various organisms play in the production of the local lesions. It is certain, however, that the lung lesions which are ordinarily attributed to these associated organisms may be produced by the Klebs-Loeffler bacillus alone.

The membrane in diphtheria is usually of a peculiar grayish-white color, and, as a rule, cannot be easily detached; in some cases, on the contrary, it may be white and easily separated. It sometimes assumes, early in the disease, a gangrenous appearance, which shows that the case is serious. Nasal diphtheria is characterized by a profuse nasal discharge, and often by a membrane. Transmission of the germ of the disease from the nose to the eye is infrequent, and does not always result in the formation of a false membrane. Frequently the pharyngeal inflammation extends through the Eustachian tube, causing inflammation of the middle ear, and in these cases a membrane may be formed. The membrane may extend downward to the larynx, causing marked dyspnœa. Below the vocal cords the membrane is not very firmly attached, and is frequite characteristic. There is apt to be a sensation of chilliness, some heightening of the temperature, and more or less pain in the back and limbs. There is nothing, however, to distinguish this stage from many other affections of children, such as simple tonsillitis. The child may often complain of discomfort on swallowing, and on examining the throat the fauces are found to be reddened. In from twelve to twenty-four hours, however, a more typical appearance will be seen in the throat. A white or grayish-white pseudo-membrane, commonly appearing first on the tonsils, develops, and on the second or third day extends to the soft palate and uvula. It may also extend backward to the pharynx. During this stage the throat becomes much swollen and the tonsils considerably enlarged, so as almost to meet at times in the median line. The diphtheritic membrane is usually firmly adherent to the mucous membrane, and, as the case progresses, assumes a brownish or yellowish-gray color, sometimes becoming gangrenous, with an extremely fœtid odor. A profuse nasal discharge in severe cases may appear at this stage, and, if the patient becomes septic, spots of ecchymoses, which are of grave import, appear on various parts of the body, and are usually significant of a fatal issue. Listlessness is present, but delirium of an active type is not common. An efflorescence resembling scarlet fever may appear in a still later stage. In addition to these lesions in the throat, the cervical glands are usually involved and become considerably swollen. The child, as a rule, shows grave constitutional symptoms. The temperature is not characteristic. It is usually not especially high, and ranges from 38.3° to 38.8° C. (101° to 102° F.). It may, however, rise to 40° C. (104° F.). A subnormal temperature is more serious than a moderately elevated one. The pulse is somewhat increased in rapidity, and is weak in proportion to the severity of the disease, but does not always correspond to the temperature; sometimes it is very slow. The slow pulse is indicative of the action of the toxin on the nervous centres, as well as to the weakness of the ventricles. Diarrhœa is a frequent but not a constant symptom. Loss of appetite, nausea, and vomiting frequently occur.

In cases of a mild type the symptoms abate towards the end of the first week, the pseudo-membrane separates, leaving a raw surface behind it, the neck becomes less swollen, and the child feels much better. It is, however, usually left in a prostrated condition for a number of weeks; and even in these mild cases the toxic effects of the disease may show themselves in the form of a neuritis with an accompanying paralysis many weeks after the diphtheria has run its course. There may also, even in mild cases, be a slight discharge from the nose, owing to the inflammation of the posterior nares. Slight albuminuria is not infrequent. A very prominent symptom in all forms of diphtheria may be cardiac weakness. In some cases the child dies suddenly without any warning, or death may have been preceded by attacks of semi-collapse. In other cases there may be a weak intermittent pulse, which continues through-may also be a purpuric condition of the skin. The picture of these septic cases is very characteristic. There is a peculiar, sweetish odor to the breath. There are cyanosis and a marked waxy pallor. There are hemorrhages from the throat and nose, with a profuse muco-purulent discharge from the latter. The cervical glands are often enormously enlarged. The membrane has been known to extend in all directions, and sometimes even through the Eustachian tubes to the external ears. All degrees of severity are met with between the mild and malignant types of diphtheria. The membrane, instead of extending upward to the naso-pharynx, as occurs in the malignant cases just spoken of, may spread downward, attacking the epiglottis and the larynx, and cause serious obstruction. The pseudo-membrane most commonly appears first on the tonsils, thence spreading to the soft palate and to the uvula. The disease may, however, begin in the mucous membrane of any part of the mouth, nose, or throat.

Nasal Diphtheria.—Diphtheria sometimes begins in the nose and spreads no farther. In these cases the disease is usually of a mild type, but it is infectious. The condition is especially liable to be overlooked, as the child for one or two days may show merely the symptoms of fever, malaise, loss of appetite, and a discharge from the nose. On examining the nose carefully, however, a pseudo-membrane will often be found. It is, therefore, very important in cases of this kind to have a bacteriological examination made, and to isolate the child until it is determined that the Klebs-Loeffler bacillus is not present. These cases are a prolific source of infection to the community at large, for even when antitoxin has been given it does not kill the bacilli, although it may stop the nasal discharge.

When the naso-pharynx is affected, either primarily or secondarily through the nares or the pharynx, the constitutional symptoms are, as a rule, marked. This is in all probability accounted for by the great mass of absorbents in the naso-pharynx, where absorption takes place so easily that general septic poisoning quickly follows. When the naso-pharynx is attacked by diphtheria, we usually meet with the most fatal results.

Laryngeal Diphtheria.—In some cases the Klebs-Loeffler bacillus produces its effects first on the mucous membrane of the larynx. In these cases the mucous membrane of the nose and pharynx may never show any evidence of a pseudo-membrane. The first symptom, as a rule, is a cough of a harsh, ringing nature. The temperature may or may not be raised. As the toxic absorption is slight, on account of the locality affected, the constitutional symptoms are correspondingly mild. The child's symptoms are those resulting from laryngeal obstruction. There is dyspnœa, with retraction of the intercostal and supraclavicular spaces, and later of the epigastrium and the lower chest. This is accompanied by an increasing cyanosis. The child is very restless, is forced to sit up in order to breathe, and, for the same reason, bends forward with its head back. In these extreme cases, unless relief is speedily afforded, the child strated bacteriologically. Although a membranous laryngitis may be due to other causes than the Klebs-Loeffler bacillus, yet this is so rare that every case of primary membranous laryngitis should be considered to be diphtheria until it has been proved that it is not. A decisive diagnosis of diphtheria in any case can only be made by determining the presence of the Klebs-Loeffler bacillus.

Recognizing that the same pseudo-membranous condition in the throat may occasionally be produced by pyogenic cocci as well as by the Klebs-Loeffler bacillus, the clinical diagnosis of a typical case of diphtheria is often difficult. A provisional diagnosis of diphtheria should be based upon the appearance of the throat of a pseudo-membrane which usually appears first on the tonsils, and has a tendency to spread to the uvula, soft palate, and pharynx. When in addition to this a nasal discharge is present, and the glands of the neck are much enlarged, a picture is presented which is not shown by any other disease. The most common difficulty met with clinically is in distinguishing between cases of acute follicular tonsillitis and diphtheria. In taking a culture, as has been pointed out by McCollom, a source of error in results is that the swab or platinum needle is rubbed over the surface of the membrane, a locality where the organism is most likely to die. The edge or, if possible, the under surface of the membrane are the proper places from which to take cultures. Cultures should also be taken from the secretions of the mouth and from the nasal discharge. Antiseptic gargles and applications should not be used a short time before the culture is taken.

Efflorescences of urticaria and various forms of erythema may follow antitoxin, and even ecchymoses. Efflorescences may occur resembling scarlet fever and measles, often so closely that the most careful examination and consideration of the symptoms are necessary, and even then the differential diagnosis is often impossible. Scarlet fever is differentiated by the presence of fever, vomiting, and the characteristic appearance of the throat and tongue; measles by the absence of catarrhal symptoms and from the fact that the antitoxin efflorescence, as a rule, first appears on the extremities. The efflorescence from antitoxin may for a few days render the patient uncomfortable, but the symptoms pass off just as an urticaria does when resulting from one of its exciting causes.

Prognosis.—Diphtheria is an extremely fatal disease, especially in the septic and obstructive cases. The mortality varies decidedly in different epidemics and according to the age. The mortality is greatest in children under two years of age, but has been lessened in cases in which the antitoxin treatment has been thoroughly used. In the acute stage death may occur either from laryngeal stenosis or by the toxin generated from the growth of the bacillus. In the later stages or during convalescence it may be due to the action of the toxin on the nervous centres. Thus paralysis of the pneumogastric nerve with obstinate vomiting and heart failure is a frequent cause of death, occurring during apparent convalescence.

The symptoms which make the prognosis especially unfavorable are the extension of the membrane to the naso-pharynx or the larynx, profuse nasal discharge, marked septic odor, extensive glandular enlargement, hemorrhage from the nose or into the skin, a high grade of albuminuria, broncho-pneumonia, and a weak heart. Morse, in an extensive study of the leucocytosis of diphtheria, has shown that it has no prognostic value. In cases of post-diphtheritic paralysis the prognosis is good, as they almost invariably recover. The prognosis in all cases of diphtheria is uncertain and should be given with caution, and no case of diphtheria should be considered benign, for at times in certain mild cases serious symptoms of paralysis may arise, and death from heart failure is liable to occur at any stage of the disease. A child who has had diphtheria is liable to suffer from the deleterious effects of the disease for months and even years. Before antitoxin was used the mortality from diphtheria in the City Hospital was 50 per cent. Since its use in a series of 4500 cases in the diphtheria wards of the Boston City Hospital the mortality has been reduced to 13 per cent., and when the moribund cases, which numbered 179, by which are meant those dying within twenty-four hours after admission to the hospital, are deducted the mortality was about 10 per cent.

PROPHYLAXIS.—All patients with diphtheria should be isolated until the Klebs-Loeffler bacillus has disappeared from the nose and throat. The time when this occurs varies from a few days to a number of weeks.

In order further to protect the community, all cases of sore throat should be examined, and if the Klebs-Loeffler bacillus is found the patient should be isolated. It is especially necessary to carry out this precaution in schools, where the conditions are so favorable for the spread of the disease.

The throats and noses of all persons exposed to diphtheria or caring for diphtheritic patients should be repeatedly examined for the Klebs-Loeffler bacillus, and if this is found they should be given immunizing doses of antitoxin, the amount and frequency of the doses to be modified as our knowledge increases. If the Klebs-Loeffler bacillus is found in these individuals, they should be isolated so long as the bacillus is present. To shorten the period of isolation, mild antiseptic gargles or douches should be employed. It is very important to keep the teeth in good condition as a prophylactic measure, as well as the mucous membrane of the nose and throat. Whether the isolation of healthy persons who have the Klebs-Loeffler bacillus in their throat or nose is advisable or not is still a mooted question. Much confusion has arisen because of the so-called pseudo-diphtheritic bacillus. The weight of evidence at present, however, goes to show that it does not exist, and that the bacteria described are merely Klebs-Loeffler bacilli of diminished virulence. At any rate, even if the pseudo-diphtheritic bacillus exists, it is so rare that it may be safely excluded in clinical work. The fact that the Klebs-Loeffler bacilli found in healthy throats may not be virulent is not an argument against isolation, because it is well known that a non-virulent form may become virulent when transferred to a different soil. Examinations of many healthy throats have shown that the Klebs-Loeffler bacillus is a very rare inhabitant of the normal throat, and that when it is present diphtheria often develops later. Theoretically, therefore, although it may be impossible or inadvisable practically, it would seem wise to consider the Klebs-Loeffler bacillus virulent until it has been proved to be non-virulent, and to consider its presence a source of danger to the community until it is proved not to be.

When an individual has been exposed to the contagium of diphtheria, immunization can be accomplished by the injection of antitoxin in doses varying according to the age. Thus, at six months the immunizing dose for infants is 300 units, and for older children 400 units. The immunization lasts for from twenty-eight to thirty days.

The length of time before a child who has had diphtheria may cease to be a source of contagion should be determined by three consecutive negative cultures from the nose and throat with twenty-four or forty-eight hour intervals.

The greatest care should be taken both by the physician and the nurse not to become infected themselves by the secretions from the mouth and nose of the patient. Especial care should be taken to avoid getting these secretions into the eyes. It is probable that with extreme care there is not much danger of the spread of diphtheria in a household, as we know its tendency is not to disseminate itself in the surrounding atmosphere, hence it is likely that with proper precautions it can be confined to the room in which the child is sick, and that if the epidemic extends beyond this room it has been carried directly by the hands or clothing of the nurse or physician.

The room should be cleared of hangings and all stuffed furniture, carpets, and clothes. The hands of the attendants should be disinfected. No handkerchiefs should be used; pieces of cotton cloth, which can be destroyed by fire, are to be preferred. Discharges should be disinfected with carbolic acid 1 to 200; mattresses, blankets, and utensils should be steamed. The floors and woodwork should be washed with corrosive sublimate 1 to 500. Books and toys should be destroyed, as there is no way of disinfecting these articles. Upholstered furniture should be immersed in boiling naphtha for two or three hours. The boiling-point of naphtha is 79.4° C. (175° F.), and is lower than the melting-point of glue. The vapor of formaldehyde is one of the most valuable germicides. General disinfection of the room is described in the disinfection of scarlet fever, page 562. Pure air and plenty of sunlight are the most effective germicides. It is important to isolate doubtful cases, especially when the symptoms are associated with a profuse nasal discharge.

TREATMENT.—The treatment of diphtheria consists (1) in attending to the hygiene and to measures directed to the general condition; (2) in the administration of remedies, either by the skin or by the mouth, to combat the toxin which is producing the constitutional symptoms; (3) in local applications to the nose, throat, or larynx; and (4) in operative measures to relieve obstruction in the larynx.

General Hygiene.—One of the most important parts of the treatment of diphtheria is the management of the room in which the patient is kept during the progress of the disease. It is known that pathogenic organisms, such as the Klebs-Loeffler bacillus, do not thrive where they are exposed to sunlight and fresh air. The room should therefore be thoroughly ventilated, and fresh, pure air should be allowed to come continuously into it. It should also be one which has a sunny exposure.

Stimulation.—In any treatment directed to the cure of diphtheria in young children, we must remember that the disease is so exhausting that the treatment, as a rule, should be forced upon the child as little as possible. Any physical exhaustion produced by the treatment is to be considered serious in young children. Diphtheria is such a depressing disease that alcohol should be given from the onset, and the amount should be relatively large. Either whiskey or brandy are valuable stimulants. For a child, one or two years old, it is well to begin with 7.50 c.c. (2 drachms) every four hours, watching the pulse and general condition. When there are indications of beginning heart failure, digitalis is indicated early in the disease, and strychnine later. When there is collapse, nitroglycerin should be given. Atropine is recommended as sometimes giving great relief when much mucus collects in the air-passages.

The following table has been arranged to show the small and large doses of digitalis, strychnine, nitro-glycerin, and atropine, which may be used at different ages:

TABLE 63.

Age.	Tincture Digitalis.	Strychnine.	Nitro-Glycerin. 1 per cent. Solution. (1 minim = $\frac{1}{100}$ grain.)	Atropine.
	Minims.	Grain.	Minims.	Grain.
3 months.	$\frac{1}{10}$ to $\frac{1}{2}$	$\frac{1}{2000}$ to $\frac{1}{1000}$	$\frac{1}{75}$ to $\frac{1}{50}$	$\frac{1}{3000}$ to $\frac{1}{1500}$
6 months.	$\frac{1}{10}$ to $\frac{3}{4}$	$\frac{1}{1500}$ to $\frac{1}{500}$	$\frac{1}{25}$ to $\frac{1}{15}$	$\frac{1}{2500}$ to $\frac{1}{1000}$
9 months.	$\frac{1}{4}$ to 1	$\frac{1}{1000}$ to $\frac{1}{300}$	$\frac{1}{25}$ to $\frac{1}{10}$	$\frac{1}{1500}$ to $\frac{1}{750}$
12 months.	$\frac{1}{4}$ to $1\frac{1}{2}$	$\frac{1}{500}$ to $\frac{1}{250}$	$\frac{1}{25}$ to $\frac{1}{5}$	$\frac{1}{1000}$ to $\frac{1}{500}$
2 years.	$\frac{1}{2}$ to 2	$\frac{1}{500}$ to $\frac{1}{150}$	$\frac{1}{15}$ to $\frac{1}{3}$	$\frac{1}{750}$ to $\frac{1}{250}$
3 years.	$\frac{1}{2}$ to 3	$\frac{1}{300}$ to $\frac{1}{100}$	$\frac{1}{10}$ to $\frac{1}{2}$	$\frac{1}{500}$ to $\frac{1}{200}$
4 to 10 years.	1 to 5	$\frac{1}{200}$ to $\frac{1}{60}$	$\frac{1}{2}$ to $\frac{3}{4}$	$\frac{1}{250}$ to $\frac{1}{150}$
10 to 12 years.	3 to 8	$\frac{1}{100}$ to $\frac{1}{40}$	$\frac{1}{2}$ to 1	$\frac{1}{200}$ to $\frac{1}{100}$

Serum-Therapy.—The treatment which, according to our present knowledge, is most efficacious in diphtheria, is essentially comprised under what is called serum-therapy.

By serum-therapy is meant the treatment of the disease by injecting into the patient the serum of an animal which has been rendered immune to the special disease which is being treated by means of inoculation with the toxin of that disease. The serum taken from the animals which have been rendered immune to diphtheria has been called the antitoxin serum. The serum can be injected under the skin. The place selected should always be one on which pressure is not exerted when lying in bed.

The dose of antitoxin serum of diphtheria should be from 1000 to 3000 units, according to the age of the child and the severity of the disease. McCollom prefers the use of from 2000 to 4000 units. The unit is the same in all reliable preparations.

The beneficial results of antitoxin are decidedly greater if the injection is made in an early stage of the disease than if later, although even when administered late in the disease it sometimes produces wonderfully curative effects. When given early, within the first forty-eight hours of the disease, even when the membrane is spreading rapidly, and inflammation of the glands with general systemic poisoning has taken place, one injection will often arrest the disease. When improvement does not take place within six or eight hours, a second dose, and if necessary a larger one should be used. Thus, if the dose was 2000 units, after eight hours a second dose of 2000 or 3000 units should be given, the latter in the more severe cases. In these very severe cases even much larger doses may be given and at shorter intervals.

The signs by which we know that the antitoxin serum has produced a beneficial result, are indicated by the improvement in the general condition of the patient, and the effect of the antitoxin on the pseudo-membrane, which is characteristic. The dose should, therefore, be repeated if the general condition and the throat do not improve in eight hours. If at the end of twenty-four hours the membrane has not begun to roll up at the edges, and the cervical glands have not diminished in size, and if a profuse nasal discharge with a septic odor is present, a third dose, and perhaps a fourth or fifth dose, may be required. When the antitoxin has produced its characteristic effect, the pseudo-membrane besides rolling up at the edges, ceases to spread, whitens, shrinks, shows a line of demarkation, and usually within the next three or four days becomes detached from the mucous membrane.

The temperature sometimes rises after the injection of antitoxin, and in a few days falls to the normal by lysis. In the more severe cases a single injection of the serum does not work so quickly. In these cases the temperature falls usually by lysis after the second or third dose, and the pulse becomes normal two or three days after the temperature has fallen. The irregularities of the pulse are not so frequent in diphtheria since the antitoxin treatment has been employed.

Antitoxin does not seem to destroy the bacilli in diphtheria, but to have some special action on the tissue-cells. If the cells have been much damaged they do not respond, and this is an argument in favor of the early administration of antitoxin. The antitoxin, however, is not able to combat complications referable to secondary infection. It is not a fact that since antitoxin has been used the type of diphtheria epidemics has been less virulent, as has been suggested.

The pains which occur during the course of diphtheria are probably forms of neuritis, and not the result of the antitoxin which has been given. The later forms of neuritis which occur two or three months after the disease has run its course have nothing to do with the administration of antitoxin. The arthralgia and efflorescences which occur during the disease may arise from the antitoxin, but are never serious, and, although the number of cases of neuritis arising in the course of diphtheria since the use of antitoxin is greater than before its use, they are rarely of a severe type, and seldom need cause any great amount of anxiety. Out of one million cases in which injections of antitoxin were given, only five deaths were reported which could be attributed to the effects of the antitoxin.

Albuminuria is one of the most constant symptoms of diphtheria. McCollom had the urine tested in two hundred cases before the administration of antitoxin, and in only ten cases was the amount of albumin increased after the administration of antitoxin, and these ten cases were of a severe type. There was no reason to suppose that the antitoxin had anything to do with the increase in the albumin. It is to be noted that, according to Baginsky's observations, nephritis may occur in mild cases of diphtheria as well as in severe cases.

Antitoxin has no influence in preventing the later symptoms of paralysis and cardiac failure, excepting as prophylactic when given early.

When there is a concurrent infection the antitoxin serum is less effectual, as it does not counteract the toxin absorption due to bacteria other than the Klebs-Loeffler bacillus. It is not safe to assume, however, that there is a concurrent affection because other bacteria are found in the throat. When the larynx is involved, with accompanying stenosis, the antitoxin serum is found to be very valuable, and has reduced the number of operative cases. The antitoxin has been found to have but little effect upon the length of time which the bacteria remain in the throat after the disappearance of the membrane.

Antitoxin should be freely used in diphtheria of the eye. The pupils should be dilated with atropine, and the eye irrigated with a 2 to 4 per cent. solution of boric acid. The child, as soon as possible, should be placed in the hands of a skilled oculist.

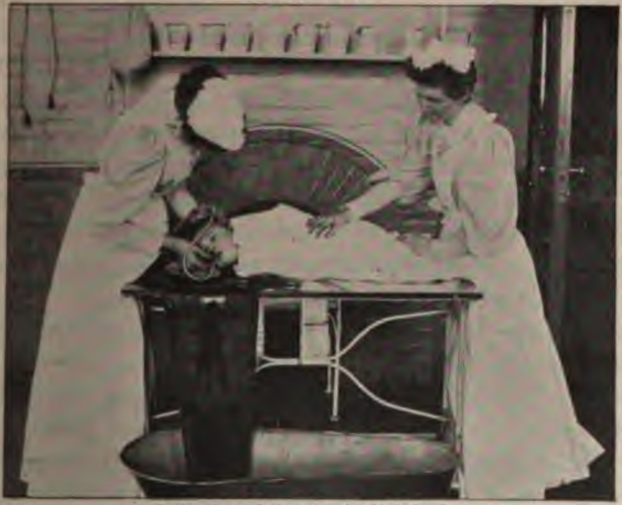
At the South Department of the Boston City Hospital, in 4500 cases, all of which had on an average at least two injections of antitoxin, no bad results were reported, and one patient had 28,000 units of antitoxin given to him and was discharged well.

In connection with the antitoxin treatment no especial drugs given internally by the mouth are indicated. Of course, symptomatic treatment of any kind is not contra-indicated.

Technique of Antitoxin Injection.—A sterilized glass syringe should be used, and the needle should be comparatively small. The part to be injected should be thoroughly sterilized with corrosive sublimate. A needle previously sterilized by boiling should be plunged into the tissues and the injection made slowly. The place of puncture should then be closed with sterilized gauze and collodion.

Local Treatment.—The local treatment of diphtheria consists in thorough cleansing of the mouth and nose with warm non-irritating solutions, such as normal salt solution or boracic acid four per cent. All strong and irritating applications to the throat in diphtheria are harmful. The technique of the local applications to the throat and nose is important. The most simple, efficacious, and safe method, and that which produces the least discomfort, is by irrigation. The same method—namely, by means of a fountain syringe—should be employed for either the throat or the nose, except that in the former a larger glass nozzle should be used than for the nose, and one which is sufficiently long to pass over the base of the tongue. The method of irrigation as employed in the Boston City Hospital and at the Willard-Parker Hospital in New York is shown in Fig. 114.

FIG. 114.



Irrigation of the nose in diphtheria.

The child should lie on its side, and the water should be made to pass up one nostril and down the other until the stream runs clear. In some cases the child prefers to sit up while the irrigation is done. Ordinarily the irrigation should be used once in two or three hours, perhaps with longer intervals at night. If the child resists this treatment it may be advisable, in order to save its strength, to omit it for a time. This rule applies to all forms of local treatment. A similar method should be used for the throat, and is of great comfort to the patient.

In the South Department of the Boston City Hospital irrigation of the throat is the routine treatment, and only in special cases is irrigation through the nose indicated. This is in order to avoid the passage of fluid infected by bacteria into the nasal end of the Eustachian tube, which might produce infection of the ear.

Considerable suffering is at times occasioned by the enlargement of the cervical glands. These are best treated with flaxseed or ice poultices. The former, however, has a tendency to produce suppuration, and the latter in most cases is the best.

When it is deemed advisable to use inhalations of vapors in stenosis of the larynx the following method may be employed, but it must be remembered that the child should not be kept in this atmosphere continuously, and should be watched carefully to see if it is speedily relieved of the stenosis; for if it is not, the continuous inhalation of steam in the comparatively small area of breathing-space which exists in the tent that is used for this purpose may of itself be detrimental to the child's recovery, from lack of sufficient oxygen. When tracheotomy has been performed an atmosphere of steam is especially valuable. Dr. McCollom's experience at the South Department of the City Hospital is that the tent need never be used, and that it is better to intubate at once.

The tent, as described by Dr. Northrup, who has used it so extensively in the Willard-Parker Hospital in New York, contains about fifty cubic feet of air. To extemporize a tent, a sheet is thrown over supports above the crib and allowed to fall over the four sides of the crib. The main point is to have a fairly large and tight enclosure. The apparatus for furnishing the steam must be free from the danger of upsetting and of setting the tent on fire. When there is a mild form of laryngeal stenosis, steam should be only moderately used, as the debility following a steam-bath is often great. Sometimes the steam will give more relief when it is medicated, but this is now considered very doubtful.

Operative Treatment: Intubation; Tracheotomy.—When the antitoxin does not relieve the symptoms of stenosis, and when the progressive dyspnœa is not quickly controlled by steam, it is well not to delay operative interference. In laryngeal stenosis operative interference is demanded by intubation or tracheotomy. The indications are marked supra-clavicular, sub-sternal, and lateral thoracic retractions lasting for two or three hours and apparently increasing in severity, a cyanotic hue of the skin, either with or without such retraction, great restlessness, dilatation of the alæ nasi, and failure of strength, even if the other symptoms are not increasing or are absent.

The following is the technique of intubation: The patient is wrapped firmly in a blanket, so that he cannot move his arms, and then placed in a horizontal position, with the head slightly raised. The mouth is held open by the gag, with its jaws resting on the molar teeth. The gag should be on the left side. Care must be taken not to have the cheek injured by the gag, and special pains must be taken to prevent its slipping. The head should be steadied by the assistant who holds the gag. The operator takes the introducer in the right hand, with the index-finger around the hook on the under surface of the handle, the loop of silk passing over his little finger, and his thumb resting on the button on the upper surface of the handle. The index-finger of the left hand is then passed down to the epiglottis, which is hooked forward; the tube is then passed into the mouth, with the handle well down on the chest of the patient; when the epiglottis is reached by the point of the tube, the handle should be given an abrupt turn, so as to bring the tube into a vertical position. As soon as the tube is well in the larynx, the button on the handle should be pushed forward, disengaging the obturator, which must now be removed, and the tube pushed into position by the index-finger. The loop of silk is passed about the ear and the gag removed. If the tube is in the larynx, the patient will immediately begin to cough with a peculiar sound, which, to be appreciated, must be heard. If the breathing becomes easier, if the cyanotic hue disappears, if the retraction of the thoracic walls diminishes, if the loop of silk does not shorten, we may be assured that the tube is in the larynx.

After becoming satisfied that the operation has been properly performed, the gag is inserted a second time, the index-finger placed on the head of the tube, and one strand of the silk loop cut, so that it can be removed. It should be especially emphasized that the finger of the operator must be a continuation of the posterior wall of the larynx, that the turn should be abrupt, and that no force must be used. If the tube is in the œsophagus, no cough will be heard; there will be no relief in the breathing; the silk loop will commence to shorten as the tube passes downward.

In certain instances intubation does not give relief, and tracheotomy must be done. If the tube becomes clogged by membrane, as is sometimes the case, it must be immediately removed. The first steps of an extraction are similar to those of an introduction. The extractor is passed into the lumen of the tube and the lever on the handle pressed so as to open the jaws, and the tube extracted by a reverse of the movements in introduction. Sometimes there is considerable difficulty in extraction, but by patience and gentleness the end can be accomplished. If the child coughs up and then swallows the tube, the accident may cause considerable annoyance to the physician, yet it is not of serious import, for experience has shown that the tube is passed by the rectum in from twenty-four to forty-eight hours, without causing discomfort. No definite rule can be given regarding the length of time that the patient should wear the tube. It is well to remove it at the end of the third or fourth day, but it is frequently necessary immediately to reinsert it. In some instances three or four extractions and introductions may be required. The most favorable cases are those in which the child coughs up the tube at the end of the third day, and does not require reintubation. Relief should come in five minutes. A child which has been intubated needs more careful watching than one in whom tracheotomy has been performed, as the accidents following intubation occur more suddenly and are more difficult for a nurse to meet.

O'Dwyer's tubes are preferably used at the South Department for the purpose of intubation.

Tracheotomy should be performed if, after intubation, relief of the breathing does not come even when the tube has been removed and replaced again. It is extremely rare for tracheotomy to succeed when intubation has failed.

Feeding.—After intubation the administration of food is often difficult, and various methods can be employed. Œsophageal feeding is the safest way of administering food, and is one of the important points to be remembered when intubation has been performed. In nasal feeding there is considerable danger of producing irritation in the middle ear, not always, however, from the Klebs-Loeffler bacillus. The method of feeding with the head lowered (Casselberry) is not devoid of danger, from the possibility of inhalation pneumonia following inspired particles of food. Nutritive enemata of peptonized milk, with stimulants, may, when retained, be an important adjunct to the treatment. Enemata, however, are not often retained. Digitalis in the enemata may be used in cases in which heart failure is present.

Treatment of Sequelæ. In the treatment of post-diphtheritic paralysis strychnine is the most valuable drug. When there is marked palatal paralysis œsophageal feeding is exceedingly important. Electricity, especially faradism, is also indicated. Secondary anæmia should be treated in the usual way, with iron, a carefully regulated diet, and by general hygiene.

INFLUENZA (LA GRIPPE).

Etiology.—Influenza is an acute, highly infectious disease. According to Leichtenstern, there are two forms of influenza. One form, which is known as *epidemic influenza*, is a pandemic disease, caused by Pfeiffer's bacillus, and characterized by great rapidity of extension, varying symptoms, a special tendency to attack the respiratory mucous membranes, an acute onset, a high degree of infection, and a tendency to become endemic during a succession of years after its first epidemic outbreak in a community.

The second form is the common endemic or *pseudo-influenza*, or catarrhal fever, the etiology of which is unknown.

Incubation.—The period of incubation is short, being usually under four days. One attack does not protect from another.

SYMPTOMS.—The onset of influenza is usually very acute, and the symptoms are variable. At times they are the same in children as in adults, but in infants and young children the symptoms are often not so severe as in the adult, although they vary in different epidemics. It is a characteristic of epidemic influenza that it has no distinct group of symptoms of its own. The symptoms are chiefly a catarrhal affection of the nose and throat, and frequently of the bronchi. These symptoms in young children are accompanied evidently by great discomfort, at times amounting to pain, in the limbs and body, although on account of the age of the patient it is impossible to determine whether much pain is present. Sometimes the only marked symptom is a heightened and irregular temperature, with marked apathy, and the disease may be so slight as to be recognizable only during an epidemic. In older children the symptoms, although, as a rule, not of so severe a type as in adults, are at times quite serious, especially if continuous vomiting occurs. Severe headache and delirium are present in some cases, and extreme emaciation, out of proportion to the fever or to the morbid conditions detectable on physical examination. Severe symptoms connected with the larynx and the lungs may also arise and rapidly disappear.

In young infants, even when no complications arise, the apathy and prostration may become extreme, and death take place seemingly from the overwhelming intensity of the infection.

In addition to the complications already mentioned, various forms of efflorescences may appear on the skin and render the diagnosis more difficult. There is a tendency to the development of tuberculosis after influenza. Relapses and recurrent attacks are not uncommon, and it should be remembered that a reinfection at times seems to take place in the sick-room. Out of a large number of cases in children examined by Schlossmann, albuminuria occurred in eight per cent., but nephritis was rare. Anæmia is a common sequela in young children. Leucocytosis in uncomplicated influenza is not present. The mental disturbances so common in adults and multiple neuritis are not marked sequelæ in children.

VARIATIONS IN TYPE OF THE DISEASE.—It is now generally recognized that there are distinct types of the disease.

Respiratory Type.—In this, the most common form of the disease, the influenza bacillus may attack any part of the respiratory tract from the nose to the pulmonary alveoli, the symptoms increasing in severity according as the latter are approached. In the milder forms there are the usual symptoms of acute coryza, fever, and headache, but these symptoms are accompanied by a far greater degree of prostration than is met with in the ordinary attacks of catarrhal rhinitis. These milder cases, without complications, may last only a few days; the temperature is usually moderate 38.2° to 39.4° C. (101° to 103° F.), but the prostration is marked. An annoying, persistent cough is commonly present.

This form is sometimes much intensified, may last longer, have a higher temperature, and show a greater tendency to complications, especially in the development of otitis media and cervical adenitis.

In another set of cases of the pulmonary type, the disease, instead of retrograding steadily, progresses, bronchitis develops, and the accompanying delirium and extreme prostration may simulate typhoid. This bronchitis has no especial peculiarities, but in young children it assumes at times a very intense grade, reaching the finer bronchi and producing cyanosis and asphyxia.

In certain instances an influenza pneumonia develops, depending either on the Pfeiffer's bacillus or on a mixed infection. This pneumonia is usually lobular, although rarely lobar. Generally, there are small areas of broncho-pneumonia diffusely scattered, and often giving rise to no other physical signs than scattered moist râles. The sputum in these cases of influenza broncho-pneumonia is never "rusty," but is muco-purulent and generally of a greenish-yellow color. A diagnosis can be made by finding the influenza bacilli in large numbers within the polynuclear leucocytes. The resolution of such a pneumonia is prolonged, ending by lysis. This class of cases is very serious, and much of the mortality of epidemic influenza arises from these pulmonary complications. The broncho-pneumonia is of the ordinary type, although possibly more irregular than in other cases.

Pleuritis may occur in the course of influenza, but is rather rare; when present, however, it is apt to become an empyema.

Nervous Type.—In certain cases there may be no catarrhal symptoms but nervous symptoms of the most varied forms. Pains in all parts of the body and limbs, cerebral symptoms simulating meningitis, convulsions, delirium, opisthotonos, symptoms simulating pneumonia without, however, the physical signs of that disease, or a typhoid condition with weak, rapid pulse, a temperature of 40.5° to 41.1° C. (105° to 106° F.), and irregularity of the heart. All these severe and alarming symptoms may pass off in two or three days and leave no trace of any of the simulated diseases, although the resulting prostration may last for a long period.

Gastro-Enteric Type.—In this form of the disease the prominent symptoms are nausea, vomiting, abdominal pain, diarrhœa, and a tendency to collapse.

The spleen is enlarged in a certain number of cases, and this, according to Osler, depends on the intensity of the fever. These symptoms may last only two or three days, and, as a rule, are not followed by an unfavorable result.

Febrile Type.—The temperature in influenza is very variable, and, as stated by Osler, may, with its accompanying prostration, be the only manifestation of the disease.

It may be remittent and associated with chills, or, on the other hand, may be of a continued type, and so prolonged as to simulate typhoid fever.

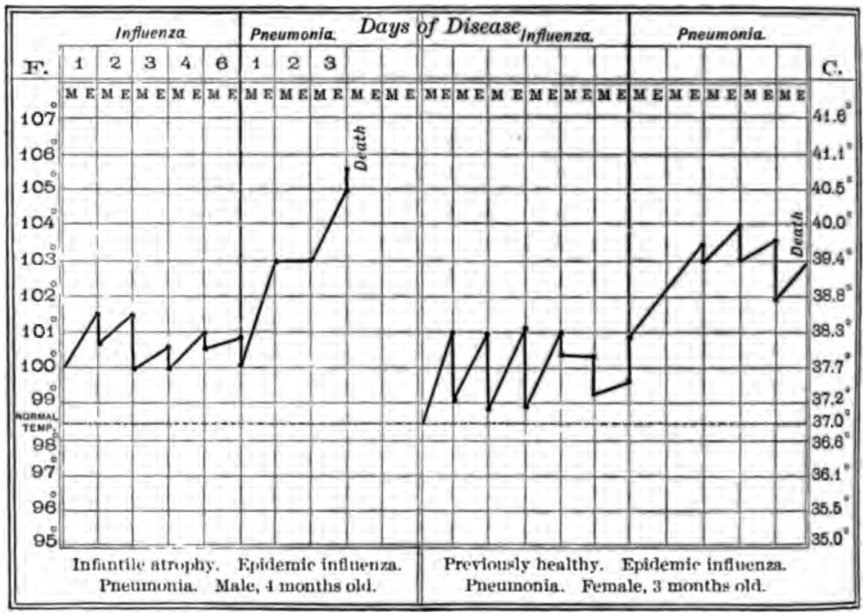
DIAGNOSIS.—The diagnosis of epidemic influenza is often difficult, unless influenza is present in the community, and is to be made by the careful elimination of other diseases, and the detection of Pfeiffer's bacillus, either in the secretions of the nose or in the sputum. The profound prostration out of proportion to the intensity of the disease is of great diagnostic value. In the early days of the disease it is often impossible to differentiate from pneumonia, malaria, the acute exanthemata, and meningitis, but later the differential diagnosis is made by the disappearance of the especial symptoms of these diseases, and the failure to find their special infecting organisms. The diazo-reaction is absent in influenza.

PROGNOSIS.—The disease in itself is not dangerous, but complications are especially liable to arise and make the prognosis much more serious. These complications are very numerous. They may be meningitis, otitis, ileo-colitis, broncho-pneumonia, and lobar pneumonia. The most common and dangerous complication of influenza is pneumonia, which is usually a broncho-pneumonia, and is of serious import, especially if the child is debilitated at the time of the attack by some previous disease. The broncho-pneumonia of influenza is of more serious import than that arising from other causes, with the exception of infections due to tuberculosis or diphtheria.

TREATMENT.—In the treatment of epidemic influenza in infants and children I have found that drugs have very little effect upon the general discomfort caused by the pain or cough. Small doses of phenacetine, 0.06 gramme (1 grain) once in three or four hours, with ten or fifteen drops of brandy, seem to yield as good results as any other mode of treatment. When there is severe and continuous vomiting, small doses of iced champagne by the mouth, enemata of bromide of potassium, and, if necessary, hydrate of chloral, are indicated. The child should be kept in bed and isolated. The bronchial and nasal secretions should be disinfected. Stimulants are especially indicated when pneumonia is present, and strychnine is valuable as a heart stimulant. A high temperature, nervous and gastro-enteric symptoms, should receive the appropriate treatment as in other diseases. When the convalescence is prolonged, especially if the case has been one of the pulmonary type, or when there is a tendency to tuberculosis or to a recurrence of the attack, a change of climate is decidedly indicated and is frequently beneficial. The diet should be milk and beef tea. Careful feeding is very important after an attack of influenza, as there is often left a tendency to infantile atrophy.

During the epidemic of influenza which occurred in Boston in 1891 I had under my care at the Infants' Hospital seven infants, varying in age from a few months to one and a half years, all of whom had epidemic influenza. Pneumonia occurred in two of the cases, and in both of these the infants died. The following charts (Charts 13 and 14) show the temperature of these cases during the course of the disease, and the rise when the infants were attacked with pneumonia.

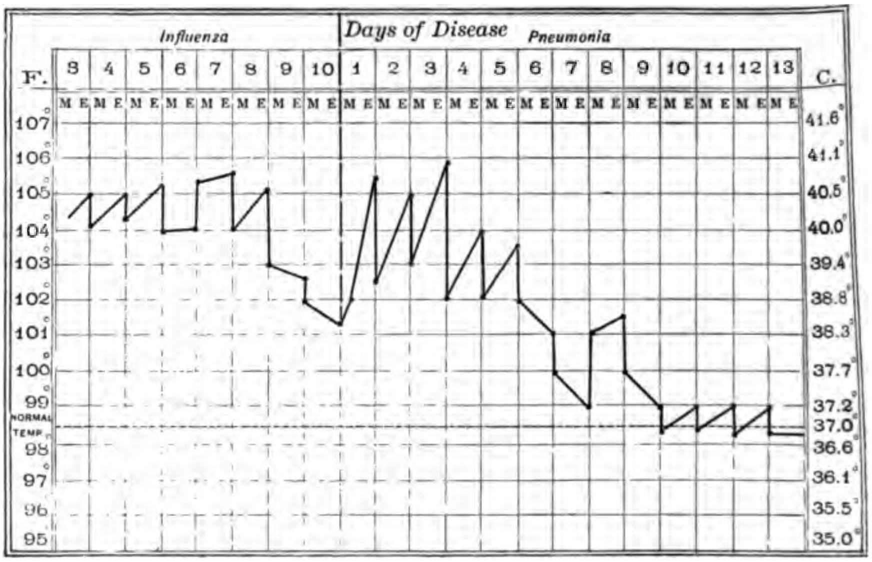
CHARTS 13 and 14.



Infantile atrophy. Epidemic influenza. Pneumonia. Male, 4 months old.

Previously healthy. Epidemic influenza. Pneumonia. Female, 3 months old.

CHART 15.



Epidemic influenza. Pneumonia. Recovery. Male, 16 months old.

Another case of influenza was complicated on the eleventh day of the disease by a lobar pneumonia.

The infant, sixteen months old, was attacked with catarrhal symptoms of the nose and throat, a slight cough, and a temperature of 40.5° C. (105° F.). The respirations were only slightly increased; the pulse was rapid. Nothing abnormal was found on physical examination. The infant was very fretful, had no appetite, cried incessantly, and seemed to have considerable discomfort. On the ninth day from the onset of the attack the temperature fell to 39.1° C. (102.5° F.), and on the following day to 38.6° C. (101.5° F.). On the evening of this day the infant, who had begun to be brighter and to notice its playthings, again seemed very sick. Its respirations increased in frequency, there was motion of the alæ nasi, and the temperature rose to 40.8° C. (105.4° F.). On the following day the temperature fell in the morning, but began to rise in the evening, and by the next day had reached 41.0° C. (105.8° F.). On this day, the thirteenth from the beginning of the attack and the third from the fresh invasion, marked flatness was detected in the right lower back, with bronchial respiration and increased tactile and vocal fremitus. This area of flatness increased, and finally involved the whole lower lobe of the right lung. On the fourth day of this new invasion the temperature fell to 38.8° C. (102° F.); on the following day it rose to 39.7 C. (103.5° F.) in the evening, and in the next two days gradually fell to 37.2° C. (99° F.). On the following day it rose to 38.6° C. (101.5° F.), and in the next forty-eight hours fell gradually to 36.6° C. (98° F.). At this time the dulness began to disappear, moist râles appeared, the infant became much better, and in a few days, although very weak, seemed bright and well, and the physical signs in the lung had entirely disappeared.

Chart 15 shows the temperature during ten days of the influenza, when a lobar pneumonia appeared and ran a course of five days, after which the temperature gradually fell to the normal point. It is possible that this case was one of pneumonia from the beginning of the attack, but it showed all the characteristic symptoms of epidemic influenza, and no flatness was found in the lung until the infant had apparently recovered from its influenza.

MALARIA.

Malaria is an infectious disease, transmitted from an infected human being by means of mosquitoes of the genus anopheles to another human being. There may be other sources of infection not yet determined.

Etiology.—The specific infection of malaria is primarily shown in the blood in the form of certain micro-organisms which, like the amœba coli, belong to the class of protozoa and inhabit the blood of the infected individual. The micro-organism of malaria has not been found in any part of the body except in the blood and within the blood-vessels, and malaria may therefore justly be said to be a disease of the blood.

The work of Ross, Grassi, Bignami, Bastianelli, Daniels, and Koch has shown that the most important and very probably the only manner of infection in malarial fever is through the bites of mosquitoes of the *genus anopheles*, which, with man, form the regular hosts of the parasite. The malarial parasite, like the coccidia, has been shown to possess both an asexual and a sexual cycle. The sexual cycle takes place in the human body and ends in segmentation, each segment attacking a new red corpuscle, and beginning again a new cycle as a fresh young parasite. Certain bodies, however, when they reach full development, do not undergo segmentation. These are the large, full-grown, sometimes wrongly called "extra-cellular bodies" of the tertian and quartan parasites, and the crescentic and ovoid forms of the æstivo-autumnal parasite. When a mosquito of the genus anopheles bites an infected human being, certain of these forms, the male elements, develop flagella in the stomach of the mosquito. The flagella attack and fecundate the female forms, which penetrate into the muscular coat of the wall of the mosquito's stomach and there undergo development. After seven days these bodies, which protrude from the external wall of the mosquito's stomach, rupture, setting free great numbers of small spindle-shaped structures, which accumulate in the veneno-salivary glands of the mosquito. When such a mosquito bites again, these bodies are introduced. Each one of these is equivalent to one of the segments of the asexual cycle, and is ready to attack a new red blood-corpuscle and pursue a similar cycle.

In this country there exist several varieties of anopheles, one of which, the *anopheles quadrimaculatus*, has been shown by Thayer and Wooley to act as an intermediate host for the parasite. It is not yet settled whether the anopheles can obtain the parasite from other sources than from man, although it seems probable that this is not the case.

The facts which so far have been accumulated in Italy and in this country appear to show that the first cases in the spring and early summer are relapses. With the appearance of mosquitoes of the genus anopheles, which occurs probably in July or August, according to the part of the country, the spread of the disease begins, the fresh cases arising as a result of the transference of the infection from relapses. The disease gradually increases during the season in which the anopheles prevails, only to diminish again with the frosts and the disappearance of this type of mosquito. The ordinary house mosquitoes, which we see in most of our cities and towns, excepting in the very malarious districts, do not belong to the harmful class. They belong to the genus *culex*. The main differences between the culex and the anopheles are readily made out. In the first place, all the common forms of culex which we see have wings free from marking, beyond the ordinary veins. All three varieties of anopheles positively known in this country have spotted wings. The most important and simple distinction, however, is the manner in which these varieties of mosquitoes sit on the wall. The culex sits on the wall with its body parallel to the surface to which it is attached and the posterior pair of legs raised over its back. The anopheles, on the other hand, sits with its body sticking out from the wall at an angle of forty-five degrees or greater, with the posterior pair of legs, which are very long, resting against the wall or hanging down. When such a mosquito is sitting on the ceiling he looks often as if he were hanging from his proboscis. This distinction is readily made out by any layman. The anopheles and culex are represented in Plate XII., opposite page 874. A third point is the difference of the mouth parts. The ordinary house mosquito, the culex, has a single long proboscis, at the root of which are two little, short, stump-like processes, the palpi. In the anopheles these palpi are of a length almost equal to the proboscis, so that on superficial examination the mosquito looks as if he had three proboscides.

The frequency of malaria in infants and young children, which has been recognized for a good while, has been strikingly brought out by Koch's studies in Java. This frequency is in all probability in part due to the well-known fact that very young children are especially subject to mosquito-bites. A rather interesting point in connection with this is that Koch uses the presence or absence of malaria in infants as an index as to whether a district is or is not malarious. In adults it is always possible that the disease may have been imported. Infants, however, have usually acquired the disease at home. The proportion of cases in infants appears to be greater than at any other age. When, then, the infants are free from malaria, or occasional cases occur among adults, it is probably safe to say that the disease is imported.

The germs of this parasite may be contained in the blood-plasma, or in the substance of the erythrocytes. The name *plasmodium* has been given to the germ found in the red blood-disks. According to Thompson, in acute paludism (malarial fever) the plasmodium is found in the form of amœboid bodies, occupying a place in a certain number of the erythrocytes or adhering to them. These bodies derive pigment (melanin) from the erythrocytes, and, after undergoing a certain degree of development, increase in size at the expense of the erythrocytes. They are found to contain this pigment in distinct granules and rods. They vary in size, and some are as large as the erythrocytes. They are at first colorless and transparent, and at the height of their development they undergo segmentation. This amœboid form of the parasite is the one commonly found in what is designated as the *tertian* variety of malaria, and is the most common of all the known forms of the parasite of malaria.

In addition to these amœboid forms, crescentic shapes of the germ, according to the investigations of Laveran, are common in the blood of certain types of paludism, irregular forms of the disease, and malarial cachexia. Like the amœboid forms, they are transparent and colorless, except for the pigment granules which they contain in their centres. They are larger than the amœboid forms, are much more rare, are much less affected by the action of quinine, and are one form of the *æstivo-autumnal* variety.

Councilman describes flagellate bodies as being most commonly found in blood which has been aspirated from the spleen; and in acute cases of malaria they may sometimes appear in other situations. They exhibit from three to eight vibrating cilia.

It is still a matter of dispute whether the plasmodium malariæ is polymorphous and thus may produce the different types of malaria, or whether there are certain distinctly separate organisms to which the name plasmodium malariæ is applied.

There is no doubt that two distinct forms of parasites of malaria can be diagnosticated by the appearance of the plasmodium in the blood, and that these two forms can be separated clinically.

Golgi is the investigator who has most clearly shown that there is more than one parasite of malaria, while Laveran is the exponent of the polymorphous theory.

The malarial parasite may be most simply and surely found by examination of a fresh unstained specimen of blood. A small drop of fresh blood is placed on a glass slide and covered with a cover-glass, making a very thin layer, and examined with an oil-immersion lens. The slide and cover must be perfectly clean and dry. Dried and stained specimens are only to be used when an examination of fresh blood is impossible.

There are definitely three varieties of the malarial parasite, *tertian*, *quartan*, and *æstivo-autumnal*. The æstivo-autumnal is usually associated with paroxysms occurring about forty-eight hours apart; these paroxysms, however, being considerably longer than those in tertian or quartan fever. Very commonly, however, owing to multiplicity of groups of parasites, the fever is irregular, remittent, or continuous. The relation of these three varieties of malarial parasites to the periodicity of the disease is shown in Table 64, on page 486.

The *tertian parasite* first appears in the red blood-corpuscles as small hyaline bodies with amœboid movements. It increases in size and develops fine, dark, pigment granules, which are in constant motion, due to the amœboid contractions of the protoplasm of the individual parasites. The red cells which contain them become slightly enlarged and gradually decolorized. Each parasite when fully grown nearly fills the red corpuscle. In the stage of segmentation, which occurs at the time of the paroxysm, fifteen or twenty segments appear, which invade fresh red corpuscles, and begin the cycle again as hyaline bodies.

The *quartan parasite* resembles the tertian, but its amœboid movements are slower, the pigment granules are coarser, darker, and less active, the full-grown organism is smaller, and the red cell in which the organism develops becomes somewhat shrunken about the parasite, and of a deeper, old-brass color. It segments into only five or ten parts.

The *æstivo-autumnal parasite* is smaller than the others and contains less pigment. The corpuscles containing them become shrunken and brass-colored. About a month after the onset of the attack, characteristic crescentic or ovoid bodies appear, which have coarse pigment granules clumped in the centre. Flagellate forms of the tertian, quartan, and æstivo-autumnal parasites may appear. The segmentation of the organism is always coincident with the paroxysms, and the interval between the paroxysms is characterized by a distinct and early stage of development of the parasites.

The tertian form is the one which is by far the most common in this part of the country, and the one which is most influenced by the administration of quinine, the other form, represented by the quartan, being peculiarly difficult to manage with quinine. In young infants the tertian form in its quotidian variety is met with most commonly. In older children, in my experience, it is the pure tertian that is most common. It will be noticed, on examining the table on page 486, that the quartan form of paludism can never represent by its intervals and paroxysms the pure tertian form.

PATHOLOGY.—There are no especial differences between the pathological lesions found in the malaria of children and those which occur in adults. According to Thayer, in *acute* cases of malarial fever, on examination with the microscope, the cerebral capillaries are found to be crowded with malarial parasites. There is usually a marked granular degeneration of the endothelium of the vessels.

The *spleen* is always enlarged, often only moderately. The parenchyma is cyanotic, of a slaty-gray color, and almost diffluent. The pulp of the spleen is found to contain enormous numbers of red blood-corpuscles, many of which contain parasites. Great numbers of white cells are also seen, some of them being necrotic. The capillaries are usually filled with red blood-corpuscles containing the plasmodia, while the splenic veins show relatively few, although they always contain large cells enclosing pigment or the remains of red blood-corpuscles.

The *liver* is swollen and has usually a slaty-gray color. The capillaries are filled with leucocytes, which contain numerous pigmented bodies. Relatively few plasmodia are found in the blood-corpuscles in the vessels. Areas of disseminated necrosis similar to those in other acute infectious diseases have been described.

The vessels of the *kidneys* contain relatively few organisms. The glomeruli may be considerably pigmented. There may be marked degeneration of the epithelium of the capsule, and at times changes in the parenchyma, especially areas of necrosis of the epithelium of the convoluted tubules. The other viscera show no special characteristic changes, except, at times, that of melanosis.

SYMPTOMS.—The symptoms of malaria as the disease occurs in infants and young children are much more varied and far more uncertain than those which we are accustomed to meet with in adults. The prominent symptom of malaria being the paroxysm, earlier authors naturally classified malaria according to the time when the paroxyms appeared, using the term quotidian when they occurred with intervals of twenty-four hours, tertian when they occurred with intervals of forty-eight hours, and quartan where they occurred with intervals of seventy-two hours. The term tertian is somewhat misleading unless we understand that it is a word derived from the Latin method of counting the day of the beginning of the febrile manifestation as the first day. The terms tertian and quartan, therefore, are simply used empirically to represent intervals of forty-eight and of seventy-two hours between the paroxysms. Again, the terms *intermittent* and *remittent* have been used commonly. The intermittent form is characterized by entire absence of fever between the paroxysms. The remittent form is characterized by the presence of more or less fever of a continued type which does not cease between the paroxysms. These terms should not be used as classifications of distinct types of malaria, as the conditions which they represent may, according to chance, appear in any of the types, and are merely caused by a variation in the behavior of the parasite. In general, in children there is a tendency in all types of malaria towards a deviation from the regular arrangement of parasites in groups, so that even in tertian and quartan fever the regularity of the manifestations appears to be not quite so striking as in adults.

According to Koplik, in pure types of paludism, either tertian or quartan, one generation of the plasmodium will be found to predominate. In the cases of tertian variety, when the paroxysms are found to be of daily occurrence, several generations of parasites, each with a different cycle of development, will be found in the blood. The same observation will be found to be true when irregular types of fever with the tertian parasite are carefully examined, and also when the blood in quartan fevers is examined. If more than one generation of parasites exists in the blood in a tertian case, the fever may become quotidian, with daily paroxysms due to the ripening of distinct sets of parasites on different days, each set of parasites taking forty-eight hours to mature. In like manner, in cases of quartan fever, through the ripening of distinct sets of parasites on different days, different combinations occur, according to the number of sets of parasites. Thus, while in the form in which there is only one parasite the intervals between the paroxysms are seventy-two hours, in that in which there are two parasites there may be an interval between the paroxysms of only forty-eight hours, and when there are three parasites there may be an interval of only twenty-four hours, thus representing the quotidian chills described by Mannaberg. The following table explains the different types of paludism as they are now understood by the most recent investigators:

TABLE 64.

The Principal Combinations of Paroxysms caused by the Plasmodium Malariæ.

	Intervals.	1st day.	2d day.	3d day.	4th day.
TERTIAN.					
Pure tertian .... (One parasite.)	48 hours.	Paroxysm.	No paroxysm.	Paroxysm.	No paroxysm.
Double tertian ...... (Two parasites. Quotidian.)	24 hours.	Paroxysm.	Paroxysm.	Paroxysm.	Paroxysm.
QUARTAN.					
Pure quartan ..... (One parasite.)	72 hours.	Paroxysm.	No paroxysm.	No paroxysm.	Paroxysm.
Double quartan ... (Two parasites.)	48 hours.	Paroxysm.	Paroxysm.	No paroxysm.	Paroxysm.
Triple quartan ...... (Three parasites. Quotidian.)	24 hours.	Paroxysm.	Paroxysm.	Paroxysm.	Paroxysm.

The table explains how the different intervals in the paroxysms are caused by the development of the parasite on different days. It will therefore be understood that it is according as the parasite happens to develop that we have a regular or an irregular periodicity. Thus, it may happen that we have two parasites, and these two parasites may develop on the same day, but at different hours. In this case, supposing that they are of the tertian type, two paroxysms may occur on the same day, followed by an interval of forty-eight hours from the time of the full development of each of the parasites until this development occurs again. In this way different broods of parasites may cause an almost infinite variety of symptoms.

The younger the individual the more likely are the pronounced chills to be replaced by some other symptom, such as vomiting, delirium, and convulsions. The paroxysms come more frequently in children than in adults and in young children a condition of apathy and somnolence, sometimes with fever, and sometimes accompanied by coldness of the extremities and a collapsed condition, very commonly replaces the chill of the adult. These symptoms, representing the onset of the disease, may often disappear as the disease becomes established, and in their place we may meet with the symptoms of some other disease, such as bronchitis, torticollis, and many other affections. The symptoms of these other diseases will often continue and be very intractable until quinine is given, when they will disappear, and thus we shall be led to believe that we have been dealing with one of the masked and misleading manifestations of the plasmodium malariæ, as illustrated by the case described on page 490.

The susceptibility of the nervous and respiratory systems in young children to produce variations in the form and type of malaria is most misleading in regard to diagnosis, the symptoms referable to a particular organ often completely overshadowing the real disease, malaria, and producing an entirely new clinical picture. The symptoms often are so indefinite and the disease frequently comes on so insidiously that the physician does not see the case until it has made considerable progress and the diagnosis is thus much obscured.

In addition to the other symptoms, severe pain in the head and sometimes in the epigastric region is met with. In the form in which the invasion is gradual, the prominent symptoms are anæmia, loss of appetite, and frontal headache of moderate type. The spleen in the majority of cases is found to be enlarged, but the well-known difficulty of detecting an enlarged spleen in young children makes it possible that in many cases there is enlargement of the spleen without our being able to detect such enlargement by percussion or palpation. Splenic and hepatic tenderness, pains in the back, extremities, and neck, and general cutaneous hyperæsthesia are occasionally observed. As the capsule of the spleen is less resistant in young children than in adults the organ seems to enlarge more rapidly and also to subside more quickly in children.

The time and character of the onset of the disease and of its paroxysms are very irregular, so much so, indeed, that it would not be practicable to dwell upon the exact differences which occur from those in the adult. In general, the fever is relatively higher, and the sweating stage is apt to be absent.

According to Holt's record of his cases in New York, the onset is acute with vomiting and prostration. The temperature is high, ranging from 40° to 41° C. (104 to 106° F.). The respiration is feeble or harsh over the back of one or both lungs, sometimes with coarse moist râles, and usually a slight increase of vocal fremitus and slight dulness on percussion. These signs and symptoms may disappear in the course of a few hours with the fall in temperature, and may recur on the following days until quinine is administered. Bronchitis was found to be the most frequent of all the complications occurring in the course of malaria, and again and again proved to be intractable until its malarial origin was discovered. Certain of these acute cases appeared to be dependent upon pulmonary congestions analogous in their pathology to the congestions of the spleen and the liver. Pneumonia, both lobar and lobular, was occasionally found as a complication of malaria. Spasmodic asthma of malarial origin was seen in some cases. These attacks were accompanied frequently by marked splenic enlargement, and were promptly relieved by quinine.

The condition of the intestinal tract varies as much as do the other symptoms. Sometimes constipation is present and sometimes diarrhœa, the latter being the more prominent the younger the child.

Subacute and *chronic* forms of malaria occur in children as in adults, characterized by anæmia, splenic enlargement, a low grade of fever, and the presence of the parasites.

DIAGNOSIS.—Malaria as it occurs in early life is far more difficult to diagnosticate by its symptoms than when the disease runs the typical course usually seen in the adult. It is the most protean disease which we are called upon to deal with in young children, and it simulates so closely so many other conditions we are likely to meet with that we should always be on our guard, and allow the possibility of the existence of the plasmodium malariæ in making a diagnosis in a doubtful case in which a periodicity is noticed in the symptoms. The only rational method of determining whether we are dealing with a case of malaria is by an examination of the blood, which at once settles the question if the plasmodium is discovered. The parasite can usually be found in a patient who has had little or no quinine, if the blood is thoroughly examined during a paroxysm by a person who has had considerable experience. The absence of leucocytosis in malaria is an important aid in the exclusion of chills and fever dependent upon septic processes.

PROGNOSIS.—The prognosis of malaria in children is good, provided that the child is removed from the malarial district and is treated with quinine. Relapses occur, even after long intervals of apparent immunity, and the disease can recur a number of times. When a child has been once attacked by the plasmodium malariæ it seems to be peculiarly vulnerable to a second attack of the organism.

PROPHYLAXIS.—The value of mosquito-nets from a prophylactic standpoint is significant, and observations in malarious districts during the last year or two strongly support this idea. The fact that a patient with malarial fever in a malarious district is a source of danger to those about him is a very important one, the deduction being, of course, that one should insist upon such an individual sleeping under a mosquito-net. The importance also of early and thorough treatment of all cases of malaria, particularly relapses in the early malarial season, is evident. In some regions destruction of the mosquito larvæ may accomplish a great deal, although much further research is necessary to determine the places in which these particular varieties of mosquito breed.

TREATMENT.—Quinine is the only drug which can be relied upon to eradicate the plasmodium malariæ from the blood, and is therefore the only medicine for this purpose which I shall mention.

The sulphate or hydrochlorate of quinine may be given to an infant under six months in doses of 0.03 gramme (½ grain); at one year the dose may be 0.06 gramme (1 grain); at two years it may be 0.12 gramme (2 grains), and it can be increased up to 0.3 or 0.36 gramme (5 to 6 grains) at five and six years. There is little danger of giving too large doses of quinine to children, as they tolerate the drug very well. The bisulphate of quinine is much more soluble than the sulphate, and for this reason is preferred by many physicians. The hydrochlorate of quinine is very much more soluble in water than either of the sulphates, requiring only thirty-five times its own weight of water as compared to eight hundred times its weight when the sulphate of quinine is used. The addition of hydrochloric or sulphuric acid renders the hydrochlorate of quinine soluble in less than its own weight of water, and the sulphate in ten parts of its weight of water. The dose of the hydrochlorate of quinine is the same as that of the sulphate. It is sometimes desirable in very severe cases to get the organism promptly under the influence of quinine. For this purpose the very soluble double hydrochlorate of quinine and urea may be given subcutaneously in doses of 0.06 to 0.3 gramme (1 to 5 grains), according to the age of the child. The latest investigations have shown that the plasmodium is most sensitive to the action of quinine when it is corpuscular. Hence the quinine should be given shortly before the paroxysm.

It has been noticed that the administration of quinine tends to interfere with the regularity of the time of the paroxysm, and in this way other variations may occur. If the paroxysm comes earlier in the day than usual, the disease is apt to be of a severe type and to be growing worse, while if the interval is lengthened and the attack is found to come at a later hour in the day than usual, it is a sign that the disease is amenable to treatment, is of a benign character, and is tending towards recovery.

The manner of administering quinine is rendered somewhat difficult on account of the bitter taste of the drug and its insolubility in water. In very young infants, and in fact in the first six or eight months of life, it is well to try the effect of quinine in suppositories. In older infants and in children it can usually be successfully concealed in a small amount of chocolate cream. Quinine should never be given in pill form, but always in capsules or in solution to make sure that it is absorbed.

The time for the administration of the quinine does not have to be regulated so carefully as in the adult. The dose should be given three or four times in the twenty-four hours, and one of the doses should be given from four to six hours before the paroxysm, so that the blood-serum shall be charged with the quinine at the time when the young and more sensitive parasites have just broken out from the blood-corpuscle. It is well to continue the treatment with quinine for some weeks after the paroxysms have ceased, as the symptoms often return if the quinine is omitted at once.

The anæmia which always accompanies the disease to a pronounced degree should be treated with arsenite of potash, or with some mild form of iron, such as the saccharated carbonate or the tartrate of iron and potash, as described under Secondary Anæmia, on page 896.

The following prescriptions, varied to suit the individual, are what I am in the habit of using in cases of malaria:

PRESCRIPTION 74.

For an Infant under Six Months.

Metric.	Gramma.	*Apothecary.*	
℞ Quininæ hydrochloratis	0·36	℞ Quininæ hydrochloratis.......	gr. vi;
Olei theobromæ.................	11·25	Olei theobromæ..............	ʒiii.
M.		M.	
Ft. suppos. no. 12.		Ft. suppos. no. 12.	

S.—One suppository to be used every 6 hours.

PRESCRIPTION 75.

For Older Children.

Metric.	Gramma.	*Apothecary.*	
℞ Quininæ hydrochloratis..........	6·00	℞ Quininæ hydrochloratis	ʒiss;
Acidi hydrochlorici dil.......q.s.		Acidi hydrochlorici dil. ....q.s.	
Aquæ destillatæ................	60·00	Aquæ destillatæ..........	℥ii
M.		M.	

Sig.—Teaspoonful once in 6 hours.

The following case of an infant, a few days old, was probably one of malaria, but no examination of the blood was made:

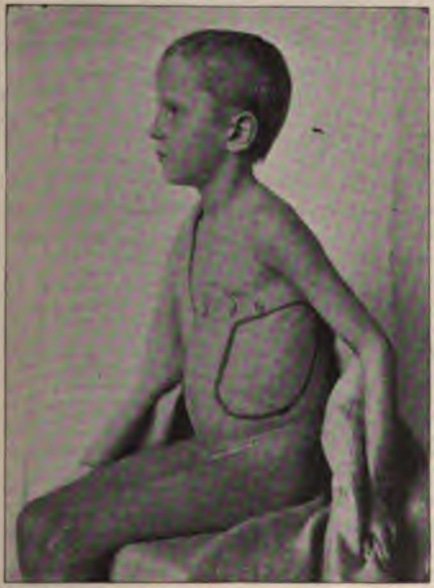
The infant's mother had malaria during her pregnancy, and some of the manifestations of the disease appeared ten days before the birth of the infant. The infant

rliest days of its life showed symptoms of severe digestive disturbance,
d by vomiting and diarrhœa, and far beyond what could be accounted for
of equilibrium of the function of the mother's mammary gland. A care-
examination failed to detect anything abnormal in its thorax or abdomen.
s of the temperature in this case, taken both in the axilla and in the rec-
d that it was of an irregular type, varying from 37.2° to 38.3° C. (99°
rectal, and that at times in the latter part of the day it rose to 39.4°
03° to 104° F.) axillary. Every day at about 1 A.M. there was a parox-
ented by cyanosis, coldness of the entire skin, both of the body and of the
collapse, and somnolence. These attacks, beginning at the seventh day of
ntil the twelfth day, when quinine in 0.03 gramme (½ grain) doses, given
ries and administered every two hours for seven doses, at once and com-
ked the paroxysms. From this time the attacks entirely disappeared, the
ell digested, and the infant seemed perfectly well.

llowing cases occurred in my service at the Children's Hospital:

nine years old, was admitted on the 13th day of February. He lived
l district until one year ago. He had had a slight cough, anorexia, malaise,
s, and rapid loss of flesh for several weeks. The movements of the bowels
irregular. According to his mother's report, he had never before had
ns of malaria. The child was pale and emaciated. On physical exami-
was resonance over both lungs, and on auscultation a few moist râles and
al sibilant râle were heard. The area of cardiac dulness and the sounds

FIG. 115.



Boy, 9 years old. Enlarged spleen. Plasmodium malariæ found in the blood.

t were normal. The liver was not enlarged, but the spleen was very
ased in size, the limits of its enlargement are marked in black in Fig.
upper border rose as high as the sixth rib in the axillary line, and extended
he left inguinal region. The urine was normal.
se represented the tertian form of malaria. The child had never had a chill

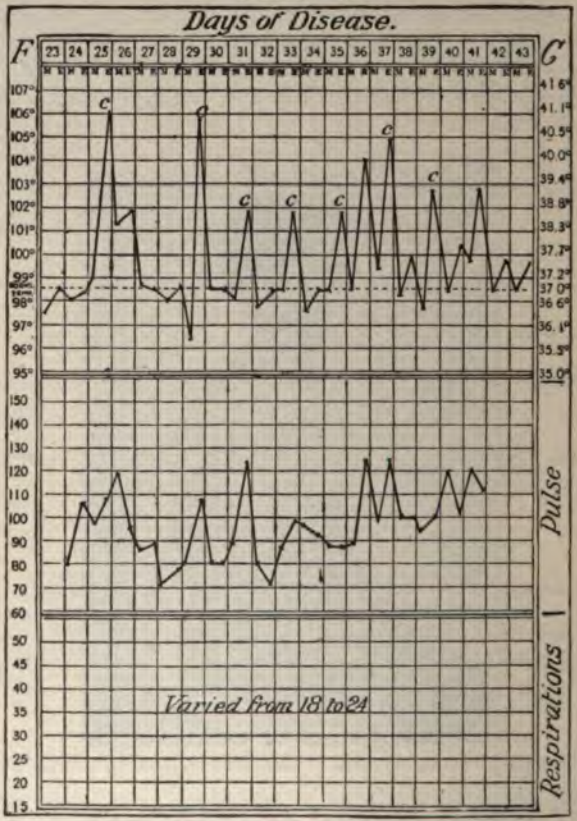
until 3 P.M. two days after entering the hospital. The chill lasted about one hour, and was followed by sweating. A paroxysm of some kind, represented either by a chill or by a decided rise in temperature with chilly sensations, occurred on the 17th, 19th, 21st, 23d, 25th, 27th, and 29th of February, March 2, and March 4, and on March 6 there was a decided rigor at 4 P.M. On March 8 the paroxysm occurred in the morning at half-past twelve. On the morning of March 10 the paroxysm occurred at about half-past eleven, and was followed by marked sweating. Between the paroxysms the boy appeared to be very well. He had a fair appetite, and gradually gained in weight and strength.

On March 10, immediately after the paroxysm, the blood showed the plasmodium malariæ. The appearance of the blood in this case is shown on Plate XII., facing page 874. The count gave the following results :

Erythrocytes	2,935,000
Hæmoglobin	36 per cent.
Leucocytes	25,500

A large number of the erythrocytes contained the plasmodium malariæ. The leucocytosis pointed towards some complication, but none was at any time discovered.

CHART 16.



Pure tertian form of malaria. (*C* means chill.)

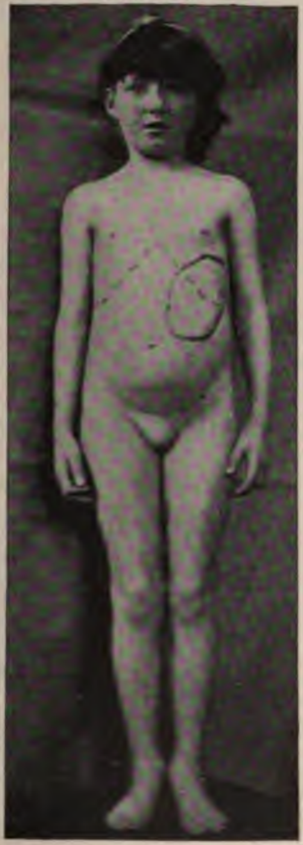
The chills continued on March 12, 14, and 16. On March 17, 0.36 gramme (6 grains) of quinine was given six hours before the paroxysm was expected to return. On h 18 there was no paroxysm. The quinine was given regularly three or four s a day for several days, and the paroxysms did not return.

Chart 16 represents the temperature and pulse of this case. The days representing disease were necessarily only approximate for the first twenty-two days, and the ild was supposed to have entered the hospital on the twenty-third day of the disease. e first chill occurred on the twenty-fifth day, as is shown in the chart.

Subsequently the quinine was omitted, the chills did not return, the spleen re-overed its normal size, the anæmia disappeared, and the child grew fat, and left the ospital in good condition.

The second case was that of a girl, nine years old, who entered the hospital also on the 13th of the month. She represented, in contradistinction to the tertian form f malaria seen in the boy, a case of the double tertian (quotidian) form. She had been living in a malarial district, but had never had any previous symptoms of malaria, although a sister living in the same house had been affected by the disease. Four weeks before entering the hospital she had an attack of vomiting, nausea, and headache, without any apparent cause. These symptoms recurred at intervals for two weeks, when she began to have chills occurring every day at about 5 P.M. These chills continued, with the exception of four days, until her entrance to the hospital.

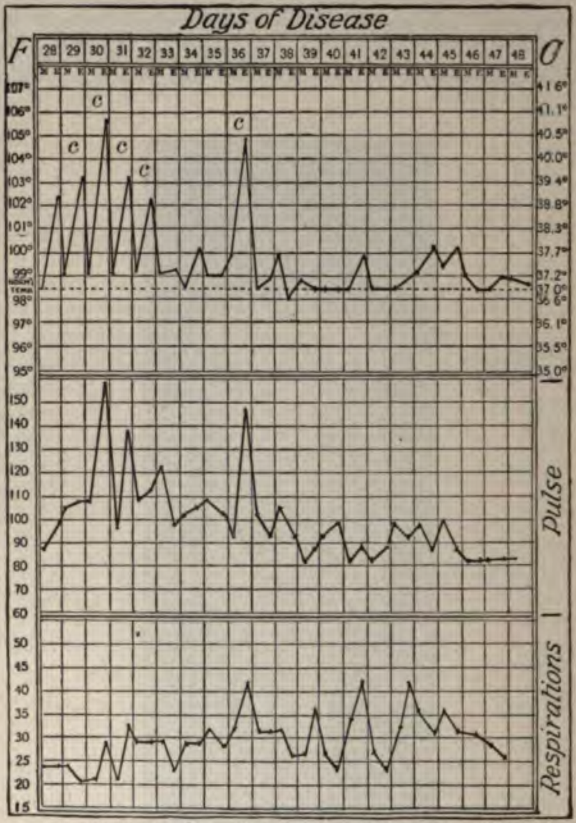
FIG. 116.



Girl, 9 years old. Enlarged spleen. Plasmodium malariæ found in the blood.

She was fairly developed and was very anæmic. On physical examination moist râles were heard over the bases of the lungs behind. The heart showed no increase in the area of dulness, but there was a soft systolic murmur over the whole præcordia. This murmur was most intense over the pulmonic area. The pulmonic second sound was not accentuated. The murmur was heard in the jugular veins. An examination of the abdomen showed it to be soft and tympanitic. The liver was enlarged, so that it extended 2.5 cm. (1 inch) below the border of the ribs. The edge of the spleen was plainly felt, and the percussion dulness extended downward to the level of the umbilicus and upward as far as the sixth rib, and is designated by a black line, as seen in Fig. 116. The urine was high-colored and had a specific gravity of 1025, but was otherwise normal.

CHART 17.



Double tertian form of malaria (quotidian). (c, chills.)

On the day of entering the hospital (the 13th) the child's temperature was raised, but there was no chill. On the following day, the 14th, there was a chill at 4 P.M. On the 15th there was a marked chill, with a considerable rise of temperature.

Immediately after the paroxysm an examination of the blood was made with the following result:

Erythrocytes	3,396,250
Hæmoglobin	30 per cent.
Leucocytes	5,000

Plasmodium malariæ present.

It was noted that the splenic enlargement was greatest during the chill.

On the 16th there was a chill, and the temperature rose to 40.6° C. (105.2° F.), the maximum attained during the course of the disease.

On the 17th and 18th the chills recurred.

On the 18th 0.36 gramme (6 grains) of sulphate of quinine was given at 12.30 P.M.

On the 19th there was no rise in the temperature, and no quinine was given.

On the 20th and 21st there were no chills, but a slight rise of temperature, and 0.12 gramme (2 grains) of quinine was given four times daily.

On the 22d she had a chill, and the temperature was 40.50 C. (105° F.).

For the next sixteen days 0.6 gramme (10 grains) of quinine was given in the course of each twenty-four hours, the spleen gradually growing smaller. As the temperature was still irregular, the quinine was then increased to 0.72 gramme (12 grains). The temperature remained normal for three days, and then was again slightly raised and irregular. Two weeks later the quinine was omitted.

The following are the records of two infants who apparently were suffering from the effects of the plasmodium malariæ, although no examination of their blood was made:

The first one was one year and ten months old. The infant had lived in a malarial district until within a few weeks of the time when I saw him.

The history which was given to me by his mother was that for several weeks he had had attacks represented by a chill or chilly sensations, occurring every day about noon. These attacks had recurred for about a week or ten days before I saw him. In connection with the chill and the fever the infant usually became unconscious, and its feet and hands were cold and clammy.

0.06 gramme (1 grain) of quinine was given to the infant on the 29th of April, and on the following day none of the usual manifestations occurred at noon, but at about 4.30 P.M. he had a chill and a slight rise of temperature, but was not unconscious. 0.03 gramme (½ grain) of quinine was then given, and on the following day, April 30, 0.06 gramme (1 grain) of quinine at 10.30 A.M. On this day there was a decided chill, and the rectal temperature rose to 40.5° C. (105° F.). During the attack the child breathed rapidly; its feet, hands, and nose became cold, and it was practically unconscious for some minutes until its circulation was restored by injections of warm water and brandy. 0.03 gramme (½ grain) of quinine was then given three times during the twenty-four hours. On the following day none of these abnormal symptoms occurred. On the next day 0.03 gramme (½ grain) of quinine was given in the morning and again at night, and this dose was continued for a few days. From this time the symptoms of malaria entirely disappeared, the infant grew less and less emaciated, became stronger, had a good appetite, and continued to thrive. No enlargement of the spleen was detected in this case.

The next infant was nineteen months old, and was brought from a decidedly malarial district.

It had previously been well until three weeks before it was brought to be treated for the following symptoms. At the time when its bath was given to it, which was between 11 and 12 in the morning, it had symptoms characterized by drowsiness and cyanosis, and it would fall asleep, and after about half an hour would wake up bright and well. These attacks, although short in duration, were very alarming and apparently serious, as, although the infant did not have any pain or convulsions, it could not be roused while in the attacks, and became so blue and cold that it was feared that it might die in one of them. At the time of the attacks the rectal temperature varied somewhat, but was usually about 38.3° C. (101° F.).

The treatment of this case was with sulphate of quinine, sometimes given by the mouth and sometimes by means of rectal suppositories. After the administration of the quinine for four or five days the attacks entirely ceased and did not return. The infant from that time continued to thrive.

TETANUS NEONATORUM.

Tetanus neonatorum is an acute infectious disease, usually occurring in infants from the third to the twelfth day of life, and is almost always fatal in two or three weeks.

Etiology.—The cause of the disease is the same as that of tetanus in the adult,—that is, the *bacillus* of *tetanus*,—and the organism is supposed usually to gain its entrance at the umbilicus. This disease is epidemic in tropical climates, but as we see it is usually of a sporadic nature.

Symptoms.—After considerable restlessness and muscular twitching, lasting for some hours, the infant assumes a very characteristic appearance. There is extreme rigidity of the legs and body. This rigidity sometimes takes the form of opisthotonos and trismus (rigidity of the inferior maxilla). The eyes are almost closed, but the infant is sleepless. The trunk and limbs are so stiff that the infant remains in whatever position it is placed. It is unable to nurse, and has a high temperature, occasionally reaching 40° C. (104° F.), and a pulse of 150 or 160. At times it will have slight convulsive attacks.

Prognosis.—It is extremely fatal. When recovery takes place the improvement is very gradual, the temperature and pulse decreasing and the rigidity of the muscles passing away very slowly, with at times a recurrence of the symptoms.

Treatment.—The treatment of this disease has thus far been very unsatisfactory. The possibility of successfully treating these cases with the antitoxin of tetanus must be considered; but the results so far obtained have not been brilliant, and it is doubtful if the cases so treated show a lower mortality than those in whom the treatment has been only symptomatic.

The form of treatment which appears to me most rational is to place the child during the continuation of the tonic spasm in a warm bath and to give it 0.06 gramme (1 grain) of hydrate of chloral every hour until the effects of the drug are shown by the lessening of the muscular rigidity and by a disposition to sleep. In addition to this treatment, small quantities of milk, 15 c.c. (about ½ ounce), should be given to the infant by means of a dropper every hour, and to each feeding three minims of brandy or some other stimulant should be added. Under this treatment a certain number of cases have been known to live.

The following case may be considered as one of the milder forms of tetanus neonatorum:

An infant, said to have been healthy at birth and to have nursed without difficulty during the first week of its life, refused to nurse, apparently from inability to open its jaws. It sometimes cried, but feebly. There were no convulsions, no vomiting, and no rigidity in any other part of the body. The temperature was not taken. On physical examination it was found that, although the infant could swallow, the jaw could not be opened wider than 1.2 cm. (½ inch). On forcing the finger between the jaws,

ıbnormal was discovered in the mouth or pharynx. The respiration was
ɔut rather shallow, and there was no evidence of injury. Nothing else ab-
as discovered.

s given 0.06 gramme (1 grain) of hydrate of chloral three or four times in
y-four hours, and two days later showed marked improvement, with the excep-
ll being unable to open the jaws widely, nothing else abnormal was discovered.
l temperature subsequently became normal, and it finally recovered.

ERYSIPELAS.

ipelas is an infectious disease, caused by the streptococcus pyo-
The term is applied to an inflammation of the skin, subcutaneous
nd mucous membranes which has the following characteristics.
ially involves the lymph-spaces and lymph-vessels. It has a
y to spread, and is attended by unusual swelling of the sub-
us tissue and an intense red color of the skin or the mucous
ne. In addition to these local appearances it is accompanied by
tional symptoms, which are mostly the result of a heightened
ture.

disease runs an acute course, is contagious, enters the individual
some abrasion of the skin or mucous membrane, and is self-

ology.—The tissues may be swollen by an accumulation of
luid, which may be nearly transparent, or turbid from admixture
s-cells. The pus-cells may infiltrate the tissues either sparsely or
masses. Sometimes vesicles are found on the surface, or there
crusts. At times more or less of the affected region is filled with
s or becomes gangrenous. In other cases, aside from the local
etechiæ are found in the serous membranes, and swelling of the
nd parenchymatous degeneration of the kidneys and liver. When
ous membranes are affected they show the same appearances as
ns of the skin, except so far as these are modified by the different
e of the tissue. The disease may attack the larynx and upper
ges and result in œdema. Pneumonia may occur as a compli-

ough the different organs, such as the spleen, kidney, heart, and
times show pathological changes, nothing characteristic of erysip-
been found in these organs, but only such changes as may occur
ontinued high temperature or as the result of sepsis.

toms.—Erysipelas may be divided into two forms,—*migrans*, ex-
from surface to surface, and *ambulans*, occurring in different parts
kin. It may also be *acute* or *chronic*. In erysipelas migrans,
s the most common form, the whole surface of the body may be
l. It is very prone to return, passing over the same surfaces of
ı again. The face and head are not so commonly attacked in
ıs in adults, and the disease seldom spreads from another part of
y to the head. When it does attack the head, it is apt to be fatal

from a secondary purulent meningitis. It at times causes great swelling and tension, and may go on to gangrene in certain localities, such as the scrotum.

The stage of the *incubation* of erysipelas lasts, according to Osler, from three to seven days.

After the first year erysipelas so closely resembles the disease as it occurs in adults that we need not consider it in this later period of life. It is a somewhat frequent disease in infants up to six months of age. It then becomes less frequent up to the first year, and after that and in childhood is rather rare. I shall, therefore, speak of erysipelas as it affects infants only.

The erysipelas of infancy may be divided into *erysipelas of the new-born* and *erysipelas of sucklings.*

Erysipelas of the New-Born.—When erysipelas occurs before the end of the third week the infant seldom lives, and indeed it is a most dangerous disease up to the end of the third or fourth month. Erysipelas of the new-born is apt to occur during an epidemic of puerperal fever. If the mother has any septic symptoms, the infant should be immediately taken away from her. I have seen a case where the mother had puerperal peritonitis following her delivery, and the infant, who was allowed to nurse her, was attacked by erysipelas.

In many cases occurring in the early days of life the disease starts on the genitals, and may be complicated by other diseases, such as empyema and especially pneumonia. During the course of the disease the fontanelle sinks, the spleen is enlarged, convulsions may occur, and peritonitis accompanied by vomiting may arise as a complication. The disease is liable to invade the tissues at any point of abrasion, whether from the forceps or from vaccination, or at the point of separation of the umbilical cord. The latter is the most common locality for the infection to take place. From this point the infection may extend and produce a gangrenous condition of the stomach or abdomen.

Although the temperature in the early hours or even days of the disease may not be raised, yet, as a rule, fever soon appears, the temperature varying from 39° to 41° C. (102.2° to 105.8° F.). Reddening and swelling, not of a high grade at first, appear on the part affected. The infants show symptoms of a general sepsis. Vomiting frequently occurs, followed by collapse and, almost without exception, by death.

Treatment.—The treatment of this severe form of erysipelas is by stimulants and a substitute food adapted to the infant's digestion.

Erysipelas of Sucklings.—When the disease occurs in the early months of life, its beginning is usually accompanied by cold extremities and collapse. The temperature is raised, and the higher its degree the graver the prognosis. The temperature curve, as a rule, is intermittent with considerable morning and evening variations, except in the more severe forms, in which there is continued high fever; in the latter cases icterus is apt to develop.

The efflorescence, although very similar to that which is seen in the adult, differs somewhat on account of the more delicate structure of the infant's skin. It begins as a faint erythema, which spreads rapidly and as quickly disappears, sometimes within twenty-four hours, and twenty-four hours later desquamation may occur. The light color of the efflorescence soon becomes darker and more intense, and is accompanied by swelling, heat, and tension of the subcutaneous tissue. After the efflorescence has continued for a certain number of days, depending upon the amount of the surface of the skin involved, the extension of the disease ceases and the temperature falls. The redness gradually disappears, and the skin becomes covered with yellowish-brown crusts. Finally, desquamation takes place, and the skin recovers its normal appearance, the disease extending over a variable period according to the greater or less extent of the surfaces invaded.

Although the disease when involving large surfaces is dangerous, yet cases in the later months of infancy recover even when the attack has been a severe one. An instance of this kind came to my notice in which an infant ten months old was attacked with erysipelas, the point of infection being the right labium.

In this case the whole vulva shortly became very tender and the disease extended to the pubes and abdomen. It invaded every part of the body and extremities and the head and neck. The eyelids and lips were the last points of attack. Even the palms of the hands and soles of the feet were affected. From the time that it appeared at one part of the body until the skin of that part assumed its normal color again was four days. When the erysipelatous inflammation extended to the feet there was marked œdema. The duration of the attack from its first appearance at the vulva to its disappearance at the eyes and mouth was about fifteen days. The infant was treated with small doses of iron and quinine, and recovered entirely.

TREATMENT.—No treatment of which I know is of any avail in cutting short the disease in early life. According to Dr. J. C. White, the extension of the efflorescence in adults can be controlled and the attack frequently aborted by the continuous application of the following wash:

PRESCRIPTION 76.

Metric.	Gramma.	*Apothecary.*	
℞ Acidi carbolici (xtals)	3 75	℞ Acidi carbolici (xtals)	ʒ i;
Alcohol,		Alcohol,	
Aquæ	āā 240	Aquæ	āā ℥ viii.

Where large surfaces are affected, the application of cold compresses tends to depress the vitality of the infant, which it is so important to sustain. During the height of the disease the infant's strength should be supported by stimulants and by the frequent administration of a food adjusted to its digestion.

Plate X., facing page 610, represents the typical efflorescence of the erysipelas of sucklings as it occurred in a female infant six months old.

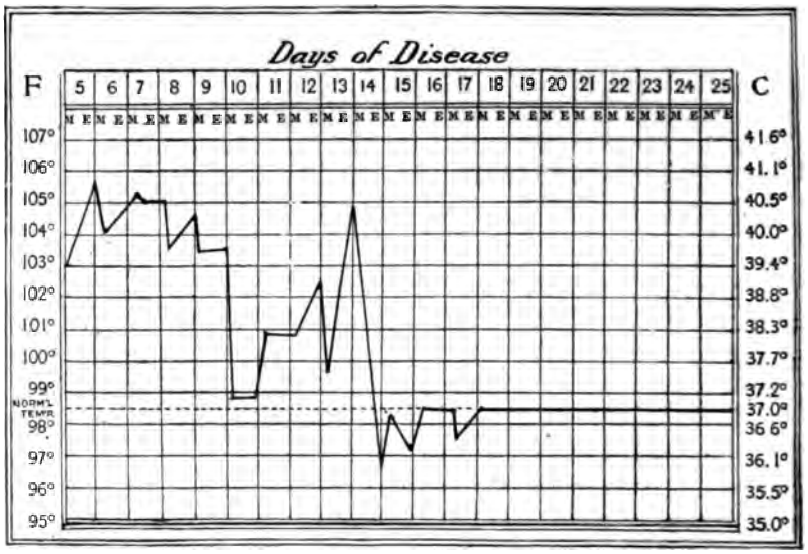
It had always been healthy, and was nursed until within three weeks of the time of entrance, when it was weaned from the mother and nursed by another woman. It was of normal weight and general development.

The first symptoms which were noticed were vomiting and a temperature of 39.5° C. (103.5°F.) in the axilla. It seemed weak and languid, looked badly, and refused to take the breast. An examination of the breast-milk showed a peculiar green color, which not only appeared in the milk when drawn from the breast, but also, when the analysis was made, appeared in the curd resulting from the precipitation of the proteids. The analysis of the milk was as follows. The nature of the micro-organism which produced the green color was not determined.

Fat	4.56
Sugar	6.36
Proteids	3.46
Mineral matter	0.13

Later in the day a pink efflorescence appeared just above the pubes, and there was found to be considerable irritation in the neighborhood of the vagina. The redness extended from the vagina to the suprapubic efflorescence. The efflorescence was of an erythematous type. On the following day it spread to the left thigh, and then to the left lower leg. The temperature continued to be raised, and the infant refused to nurse. Small quantities of a substitute food with the following percentages, which had to be varied from day to day, were given to it:

CHART 18.



Erysipelas of legs. Female, 6 months old.

PRESCRIPTION 77.

Fat	2.50
Sugar	6.00
Proteids	1.50

There were no convulsions or other symptoms, but the infant lost somewhat in strength and weight and its face looked pinched.

The efflorescence on the left leg began to fade on the ninth day of the disease, and on the tenth day the temperature became almost normal. On the following day, however, it again rose, and a fresh efflorescence began to appear on the right thigh continuous with the efflorescence of the suprapubic region. This efflorescence extended down the right leg to the ankle.

On the twelfth day the skin of the left leg was in some places almost normal, in others was covered by thin brownish-yellow crusts. The suprapubic region and the right leg as far as the ankle were covered with a bright red efflorescence sharply bounded by normal skin below, just above the ankle, as though it were a stocking. The whole leg was swollen, was hotter to the touch than the sound skin, and presented a somewhat raised, glistening appearance.

No external applications and no drugs were employed in this case. The milk was carefully modified, and small doses of brandy were given.

A few days later the temperature became normal, the efflorescence began to fade, desquamation subsequently took place, and the skin finally recovered its normal appearance. The infant gradually regained its strength, became perfectly well, and had no return of the disease.

Chart 18 shows the temperature during the course of the erysipelas in this case.

This form of erysipelas may become chronic, and this is more apt to occur in children than in infants. It is also most common in children who are in a debilitated condition, and may occur at intervals of three or four years. It is in older children frequently connected with chronic inflammations of the Schneiderian membrane, and in these cases is peculiarly intractable to treatment.

AMŒBIC ILEO-COLITIS.

(*Synonyms.*—**Tropical or Endemic Dysentery**).—ETIOLOGY.—The later researches of Flexner and others seem to point towards the fact that there are at least two forms of tropical dysentery. One form, the bacillary, is as yet *sub judice*, while the other, the amœbic, has been established as a specific infectious disease, caused by a well-recognized organism called the amœba coli. The amœba coli is an animal micro-parasite belonging to the group of protozoa. The disease is of rare occurrence in children, and is very infrequent in northern climates. It is seen for the most part in the tropics. A common source of infection is by means of drinking-water.

PATHOLOGY.—Amœbic ileo-colitis has its own definite anatomical lesions, which consist of round, oval, or irregular ulcers, the edges of which are infiltrated and undermined, so that individual ulcers may be connected by submucous sinuses. Amœbæ are found in the lesions and in the intestinal discharges. The large intestine is more commonly the seat of the disease, especially the cæcum, hepatic and sigmoid flexures, and the rectum.

The disease may advance by progressive infiltration of the connective tissue of the intestines, giving rise to large sloughs. In the same manner the liver may become infected, producing lesions in the nature either of local necroses of the parenchyma or of abscesses, which may be single or multiple. Cultures taken from the abscesses are generally sterile, but the amœbæ may be in the walls of the abscesses. Similar abscesses found within the lungs are a result of direct extension from the liver through the diaphragm.

Symptoms.—The disease is usually acute in its onset, but sometimes it may be gradual. The duration may be two or three months. The diarrhœa at first may be transient and trifling in character, but in the severe gangrenous forms is abrupt and intense. The course of the disease is irregular. The diarrhœa is characterized by exacerbations and remissions, and progressive loss of flesh and strength. The fever is moderate, and in some cases is absent. Nausea, vomiting, abdominal pain, and tenderness are usually only present in the severer forms. The stools present a variety of appearances; they are generally watery and mixed with mucus, and sometimes with blood, but in the intermissions and convalescence are formed. The amœbæ, with their characteristic movements, may be found when the stools are fluid, but rarely if they are formed.

Diagnosis.—There are no especial symptoms by which to distinguish this form of ileo-colitis from other forms of ileo-colitis, which are described on page 820, and the only positive proof of the existence of the disease is the presence of the amœbæ in the discharges.

Prognosis.—The prognosis is very unfavorable. Recovery is slow. The anæmia and weakness delay convalescence, and relapses are common. An involvement of the liver generally leads to a fatal issue.

Treatment.—The treatment which has been followed by the most favorable results is, in addition to frequent and thorough irrigation of the intestine, injections of solutions of sulphate of quinine (1 to 5000). This treatment, however, affects only the amœbæ which are in the intestine, and not those which are embedded in the tissues. The anæmia should be treated as described on page 896. The diet should be carefully regulated and adapted to the age of the individual. Milk, beef-juice, egg albumin, and broths should constitute the principal food, and in the convalescence it is necessary to exercise especial care in the management of the digestion.

CHOLERA INFANTUM.

Cholera infantum is an infectious disease caused by a specific organism not yet discovered, and characterized by acute gastro-enteric disturbance with intense choleriform symptoms. The term cholera infantum should be exclusively restricted to this class of cases, and should not be used to designate the many acute and serious attacks of vomiting and diarrhœa which are so often designated cholera infantum. It is a rare disease.

Etiology.—The organism which has been found most commonly in cholera infantum is in the proteus group. The disease occurs in the first two years of life, and in its development is probably closely associated with the food, for it has been noticed that infants who are fed exclusively on pure and sterile foods, such as breast-milk, are not liable to be attacked by it. It is also significant that the disease occurs only in hot weather.

PATHOLOGY.—The pathology of cholera infantum has not yet been satisfactorily determined, but it seems to be a non-inflammatory disturbance of the whole gastro-enteric tract, without any gross lesion beyond a desquamative catarrh, and sometimes hyperæmia of the mucous membrane.

SYMPTOMS.—The onset of cholera infantum may be sudden, but, as a rule, it is preceded by some form of gastro-enteric disturbance, which, by causing an irritation of the mucous membrane, renders the infant vulnerable. When, however, the disease has once gained a foothold, the development of the symptoms is very rapid.

After a variable but generally short period of restlessness and apparent abdominal discomfort, the infant begins to vomit. The vomiting is either accompanied or quickly followed by profuse diarrhœa. After the stomach and intestine have been emptied of the food which may happen to be in them at the time of the onset, the vomitus and the diarrhœal discharges are chiefly serous; and it is this watery consistency of the discharges which especially characterizes the disease. As a rule, the discharges are odorless, and consist of serum mixed with epithelial cells and many bacteria. Although the disease is more likely to attack weak and debilitated infants, yet it often attacks those who are healthy and robust. It may run its course to a fatal issue in from twenty-four to forty-eight hours. The extremities soon become cold, the skin is pallid or even cyanotic, and the face pinched. The abdomen may be a little distended, but is soft, and soon becomes rather retracted. The pulse is rapid and difficult to count. The respirations are somewhat quick and superficial. The temperature of the entire surface of the body is low, but the deep rectal temperature is high, 39.4°, 40°, or 40.5° C. (103°, 104°, or 105° F.). The thirst is great and is a very prominent symptom. The fontanelle very soon becomes depressed. The urine is very scanty and sometimes suppressed. It is concentrated, highly acid, almost always contains albumin, and often casts and blood, and nervous symptoms, such as twitching of the arms and great restlessness, are present. Rapid emaciation takes place, and all the symptoms increase in severity. At first the infant whimpers, but soon it becomes listless, falls into a stupor, or may have convulsions. The infant may die in this stage, which closely resembles the algid stage of cholera Asiatica. The disease appears to be self-limited, and if the infant survives the first two or three days a crisis comes, the skin becomes less cool and of a better color, the vomiting and diarrhœa grow less frequent, and finally it is left with a slight amount of simple diarrhœa and occasional vomiting. These symptoms may become chronic, in which case the infant finally dies of exhaustion or from an attack of one of the other gastro-enteric diseases, to which it is left very susceptible.

DIAGNOSIS.—The diagnosis of cholera infantum is not difficult if the characteristic symptoms are borne in mind; these are rapid onset, constant vomiting, frequent serous discharges, intense thirst, high rectal temperature, low surface temperature, collapse, depressed fontanelle, sudden loss of weight, and distressed, restless expression, suggesting speedy death, all developing in from twenty-four to forty-eight hours.

Prognosis.—The prognosis is bad. The more violent the attack, the higher the temperature, the less the vitality, and the warmer the weather, the worse is the prognosis. When the infant has survived the very acute symptoms which appear in the first two or three days, the prognosis is much more favorable.

Treatment.—Cholera infantum is so formidable in its attack that it must be treated most energetically if we hope to succeed in saving the infant's life. The indications for treatment are (1) to assist the effort which nature is making to free the stomach and intestine from the poison which is in them; (2) to restore the surface circulation, which is so seriously interfered with; (3) to supply water to the tissues, which are being drained to so grave an extent; and (4) to support the strength until the disease has run its course.

The poison seems to act with especial virulence on those portions of the economy where it is most concentrated,—namely, the stomach and the intestine. We therefore have at first extreme irritation of these parts, which causes increased peristalsis, and later vasomotor paralysis, with great transudation of serum. This condition of the gastro-enteric tract is to be especially borne in mind during the whole course of our treatment.

In this disease we should not attempt to use any remedy which works slowly. The condition of the mucous membrane is in all probability such that absorption of drugs does not take place readily. The administration of drugs is, therefore, contraindicated, for they may later, when absorption is being restored, prove fatal by their cumulative action. During the acute stage of the disease the digestive functions fail to act, and therefore food of any kind will be only an additional source of irritation.

Early in the attack, and when the vomiting has not caused much prostration, the stomach should be thoroughly washed out with warm water and the intestine should be irrigated. If the rectal temperature is very high, ice-cold water may be used for irrigation. When the vomiting has continued for some time and there is prostration with great thirst, the infant should be allowed to suck sterilized ice-cold water from the bottle. At first nothing else should be given by the mouth.

The infant should be placed at once in a warm pack. This should be done by wrapping it to the chin in sheets wrung out of water at least as hot as 38° C. (100.4° F.). It should then be enveloped in a hot blanket. This procedure should be repeated as often as the infant shows signs of collapse or much cyanosis and coldness of the skin. This is the best method that I know of to restore the surface circulation. In extreme cases the subcutaneous injection of normal salt solution can be tried. Care should be taken not to introduce in fifteen minutes more than 37.5 c.c. (1 drachm) for every pound of the child's weight.

While the infant is in the hot pack, water can be given freely by the mouth, and, if necessary, small and frequently repeated doses of stimulants, unless they appear to excite vomiting, in which case they should be given hypodermically.

If the vomiting and diarrhœa continues to be excessive after this treatment, small doses of morphine, 0.0006 gramme ($\frac{1}{100}$ grain), and atropine, 0.00008 gramme ($\frac{1}{800}$ grain), for an infant a year old, can be tried hypodermically. The effect should be carefully watched, and the dose repeated if necessary.

If, after the vomiting and diarrhœa have ceased, the heart's action continues very weak and does not respond to stimulants, small doses of digitalis should be given. The greatest caution should be employed in using drugs, however, as they generally do more harm than good.

If an absolutely fresh and sterile milk can be obtained, it can be used as a food, as in any of the other forms of gastro-enteric diseases which I have already described, but for some days the percentages of the elements in the milk must be much lessened, and the child's strength must be supported mostly by stimulants freely diluted with sterilized water.

CHOLERA ASIATICA.

Cholera Asiatica is a highly infectious disease, caused by the comma bacillus of Koch, which manifests its most violent symptoms in the gastro-enteric tract. Its symptoms very closely resemble those of cholera infantum. The disease in infants should be diagnosticated from cholera infantum by finding the comma bacillus in the vomitus or in the discharges. There are no especial differences between cholera Asiatica in the adult and the same disease in the infant. It is exceedingly fatal during infancy and childhood, and young infants who are attacked by the disease during a cholera epidemic seldom live. The treatment is the same as that which has just been described for cholera infantum.

PERTUSSIS.

Pertussis (whooping-cough) is a highly infectious disease, affecting the respiratory tract, characterized by periods of spasmodic coughing, succeeded by a prolonged inspiration and accompanied by a peculiar sound called the "whoop."

Etiology.—The cause of pertussis is probably a micro-organism, but this organism has not yet been definitely determined.

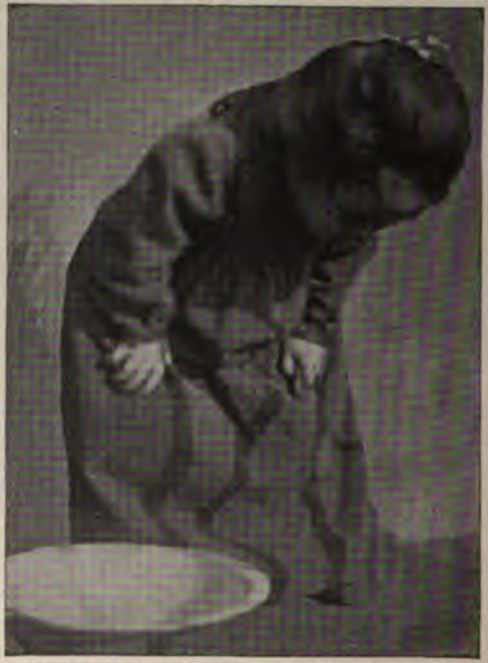
Zusch found in twenty-five cases peculiar small bacteria in the sputa. They were sometimes seen in clumps, or masses, or in leucocytes. They were more numerous in uncomplicated cases, while when marked bronwhoop which characterizes the disease appears. The cause of these paroxysms seems to be a spasm of the larynx. This is accompanied by a feeling of suffocation. The paroxysm begins with a number of short, spasmodic, expiratory coughs, succeeded by a long-drawn inspiration and by the peculiar whoop. During the paroxysm, especially in severe cases, the face and mucous membranes become cyanotic, the eyes protrude, the conjunctivæ are congested, and the child looks as though it would die of asphyxia. After a few seconds the child, with a convulsive cough, expels some tenacious mucus, and is then relieved, or the attack returns again, and again subsides, and the symptoms of asphyxia pass away. These paroxysms are often followed by vomiting. They may occur only four or five times in the twenty-four hours, or again much oftener,—at times thirty, forty, or fifty times. At the onset of the attack the children are usually very much frightened, and either run to the mother or nurse for aid, or go to some part of the room where they can be undisturbed during the attack. In certain children, after the severe paroxysms have lasted for some time, a small ulcer is formed on the frænum of the tongue, from being driven against the edge of the lower teeth during the paroxysms. During the course of the paroxysmal stage of pertussis it is quite common to have subconjunctival hemorrhages; rarely deeper-seated hemorrhages take place in the meninges and in the deeper parts of the eye. In protracted cases petechiæ sometimes appear in the skin. Epistaxis may also occur.

Examinations of the chest during the attack have shown that the pulmonary resonance is lessened during the expiratory stage and is clear during the prolonged inspiration. The auscultation usually shows diminution or absence of the respiratory murmur. Bronchial râles are heard occasionally.

Koplik has noticed an increase in the area of the relative cardiac dulness during the paroxysmal stage of pertussis, which is often accompanied by a slight blowing murmur limited to the apex of the heart. This may well occur from the engorged condition of the right side of the heart, which subjects the heart to a great strain and may thus result in dilatation. The heart-sounds are apt to be irregular during the paroxysm, and in protracted cases during the intervals the pulse is often irregular and accelerated, while the respirations are not especially increased unless some complication has arisen. In severe cases of pertussis the kidneys are sometimes congested, as shown by the appearance in the urine of albumin, casts, and blood-cells. Sugar has also been frequently found.

After the disease has lasted for some weeks there is usually a certain amount of œdema of the face, especially under the eyes. The paroxysms are precipitated by nervous excitement or by an irritation in the throat or the respiratory tract, such as may result either from swallowing or from the inhalation of dust. The stage which is accompanied by the whoop and the more exaggerated paroxysms commonly lasts for three or four weeks or even longer. The paroxysms then become less severe, and, although the cough continues, the whoop gradually becomes less frequent, and after three or four weeks ceases entirely. When uncomplicated, the duration of the disease is usually three or four months. Slight changes in the atmosphere or exposure will give rise to a relapse. The relapses, however, are not, as a rule, of a severe type, and in these cases the cough seems to arise from renewed irritation of the sensitive mucous membrane of the respiratory tract rather than from a fresh infection by the specific germ. A persistent cough following an attack of pertussis may sometimes be caused by an insidious form of broncho-pneumonia.

Fig. 117.



Pertussis during paroxysm. Female, 4 years old.

Fig. 117 represents a child during a paroxysm of coughing in pertussis. She had just begun to cough, had become decidedly cyanotic, and was aiding the expiratory effort by bending forward and placing her hands on her knees at the moment the photograph was taken.

The period of infection is supposed to last for a certain time after the whoop has ceased, and if the cough continues it is well to allow for a period of infection of three weeks after this cessation. It is possible, however, that the whoop may occasionally occur for long periods after the child has ceased to be a source of infection to other individuals.

Complications.—The complications which arise in pertussis are usually of a grave nature. The dangers from hemorrhages, unless in the form in which they occur in the meninges, are not great. The complication of broncho-pneumonia is very serious, and often fatal. The especial susceptibility of these cases to the development of some form of tuberculosis, especially a tubercular broncho-pneumonia, renders the stage of convalescence or decline one which demands most careful observation. Severe and even fatal emphysema may occur in pertussis.

Convulsions may arise not infrequently in infants and end fatally. They are usually caused by general reflex disturbance, by cerebral congestion, or by some cerebral lesion. Spasm of the glottis may also very rarely cause death in greatly debilitated children. Excessive and obstinate vomiting at times becomes a serious complication, and may reduce the child's strength to a point which often gives rise to a doubt as to its recovery. It is an especially grave complication in infants who are already much debilitated. Grave intestinal complications are liable to occur, especially in the summer, and a pronounced atrophic condition is not uncommon when the disease is at all prolonged.

DIAGNOSIS.—The diagnosis of pertussis cannot, as a rule, be made until the child whoops. Sometimes, however, when another child in the family has undoubted pertussis, a spasmodic cough may allow the diagnosis to be made before the whoop has developed. It is probable that a child may have pertussis without at any time developing the whoop. In doubtful cases an examination of the blood in reference to the presence of an increased white blood count consisting largely of lymphocytes may aid in making the diagnosis.

In some children a simple catarrhal laryngitis will simulate pertussis quite closely; but, although in these cases there are paroxysms of spasmodic coughing, a pronounced whoop does not occur, and the symptoms do not progressively increase and last for a long period. The diagnosis of pertussis can usually be made by the swollen aspect of the face, the paroxysmal cough followed by the expulsion of tough mucus and vomiting, and the long duration of the attack.

PROGNOSIS.—Pertussis is a very serious affection in young infants, and also in older children who are debilitated or poorly cared for. When it is complicated it is one of the most fatal diseases which occur in early life. When it occurs in older children the prognosis is favorable, provided that they have previously been well and strong, that they are well cared for, and that no complications arise.

In some cases young infants, if their vitality is unusually good, and if they are carefully nursed and made to take a sufficient amount of food, show remarkable powers of resistance during attacks of pertussis.

A case of this kind that came under my care was that of an infant five months old. In March she suffered from an attack of epidemic influenza, which lasted about twelve days, and from which she finally recovered. She was then attacked with measles, and after the temperature had fallen to the normal point she was taken with pertussis. After two or three days the cough increased in severity, and after two weeks the infant began to whoop. The attack lasted for two months, and she finally recovered. During the whole course of the disease she took over 600 c.c. (20 ounces) of modified milk in the twenty-four hours, and for a short time small doses of brandy were given. No drugs were administered. The temperature varied from 37.2° to 37.7° C. (99° to 100° F.).

Prophylaxis.—Pertussis is so highly contagious a disease, and may be so serious an affection in certain children, that a rigid prophylaxis should be enforced. It is the duty of those who take care of children with pertussis to see that they are isolated during the whole course of the disease.

Treatment.—In the treatment of pertussis we must take into consideration the age of the individual, the stage of the disease, and the presence or absence of complications. In the early months of life, after the disease has lasted for a week or ten days and has become more severe, the infant will usually show symptoms of general circulatory disturbance. The great strain thrown upon the heart during the paroxysms quickly affects the general strength of the infant, a marked interference with its nutrition soon appears, it loses in weight, and often refuses its food. At times it will become somewhat cyanotic even between the paroxysms, and there is danger not only from the severity of the paroxysm, but also from the vitality of the infant, which may be so much interfered with as to prevent its recovery. In cases of this kind the nursing is of the utmost importance. The infant should never be left alone, should always be taken up whenever a paroxysm is approaching, and should be assisted in appropriate ways until the paroxysm is over. Holding the infant in different positions, sometimes bending the head and body forward at the end of the paroxysm so as to aid by gravity the expulsion of the tenacious mucus, is desirable. At times, also, the finger covered with a thin cotton cloth can be quickly introduced into the throat and the mucus withdrawn in this way. It is of the utmost importance that the infant should be surrounded continually by fresh air. For this purpose two rooms should be used, if possible, one of which should have all the windows thrown wide open, so that the air can be completely changed before the infant is brought into it, and the patient should be alternately taken from one room to the other, the temperature of the rooms being kept as equable as possible.

The nutrition of the infant is so easily affected that the utmost attention should be paid to the administration of the food. Small quantities of a milk carefully modified to suit its digestion should be given at frequent intervals, preferably after the occurrence of a paroxysm, as it is then more likely to retain the milk in its stomach a sufficient length of time for it to be absorbed before the next attack. The amount of food which the infant retains in the twenty-four hours is an important factor in the treatment. In infants of from six to twelve months at least 600 to 750 c.c. (20 to 25 ounces) of milk should be taken and retained in the twenty-four hours. When the amount is lessened to 360 or 450 c.c. (12 or 15 ounces), the infant's nutrition, as a rule, suffers to such an extent that unless this amount can be increased a fatal issue is likely to result.

Stimulants, in the form of brandy or whiskey, should be given early in the attack. When the cyanosis is a prominent feature and the pulse is irregular and intermitting, small doses of digitalis should be given, adapted to the age of the child, as in Table 64, on page 486. In these cases, also, the administration of oxygen is a valuable adjunct to the treatment. At the height of the attack, when the paroxysms are severe and especially frequent at night, the burning of cresoline in the room at night is sometimes beneficial. In the milder forms of the disease which are not accompanied by the more severe symptoms just enumerated, belladonna or atropine often proves valuable. There is no drug, however, which is a specific for pertussis at any age.

For older children whose health has previously been good, there is no especial treatment, except that they should have as much fresh air, free from dust, as possible, and that food should be given them after they have vomited.

When complications arise, the treatment is that of the complicating disease.

In cases which are protracted, a change of air, either to the country or to the sea-shore at suitable seasons, is often followed by an apparent shortening of the duration of the attack.

In some cases, even in older children and where no complications are present, the attack of pertussis may be so severe as to prove serious. Northrup reports the case of an infant, under one year of age, who died during a violent paroxysm in pertussis, and in whom the autopsy showed extensive vesicular emphysema with great distention of the walls of the alveoli. The following case occurred in my practice:

A boy, six years old, had had pertussis for five weeks. For two weeks previous to my seeing him the cough had been so frequent and so constantly accompanied by vomiting that the child had been unable to retain any food. He was very much emaciated, and was so weak that he could not stand. This condition lasted for a week or ten days: he then began to improve, and finally recovered entirely. There were no complications.

ACUTE INFECTIOUS OSTEOMYELITIS.

Acute infectious osteomyelitis is an acute arthritis attended by suppuration at the joints. It has been described under various other names, such as *acute arthritis of infants*, *acute purulent synovitis of infants*, *acute epiphysitis*, and *pyæmia of the bone*.

Etiology.—Nichols has shown that acute infectious osteomyelitis is an acute inflammation of bone due to any one of a number of pathogenic organisms, hence osteomyelitis is not a specific disease, but belongs to the group of septic pyæmias. The staphylococcus pyogenes aureus is the organism that most commonly produces the disease. An early period of development and the exanthemata predispose to osteomyelitis. Traumatism must be recognized as an exciting cause.

Pathology.—In infants the disease commonly originates from a septic infection of the umbilicus or pharynx. The process attacks the bone-marrow primarily, and the joints become secondarily involved if the process originates or extends to the end of the bone and is not limited by the shaft. The joint infection may come either from separation of the epiphysis, which is wholly or in part intra-capsular, or from a growth of pyogenic organisms along the blood- or lymph-channels of the epiphysis, frequently setting up an osteomyelitis of the ossified portion of the epiphysis and spreading thence into the joints. The two affections, therefore, represent different phases of bone infection, rather than different diseases of the bone. The morbid process sometimes involves the entire structure of the bone, including the periosteum.

Osteomyelitis begins as a hyperæmia of the bone-marrow. Later, suppurative foci of a dull-yellow or grayish color appear, while in severe cases the entire marrow becomes purulent and the Haversian canals of the cortical portion become filled with pus. Metastatic abscesses and thrombosis of the veins of the marrow may follow. When the infection enters the joint the cartilage is softened and destroyed and an abscess is formed. Osteomyelitis may, however, be the cause not only of a suppurative but of a simple inflammation of the joint.

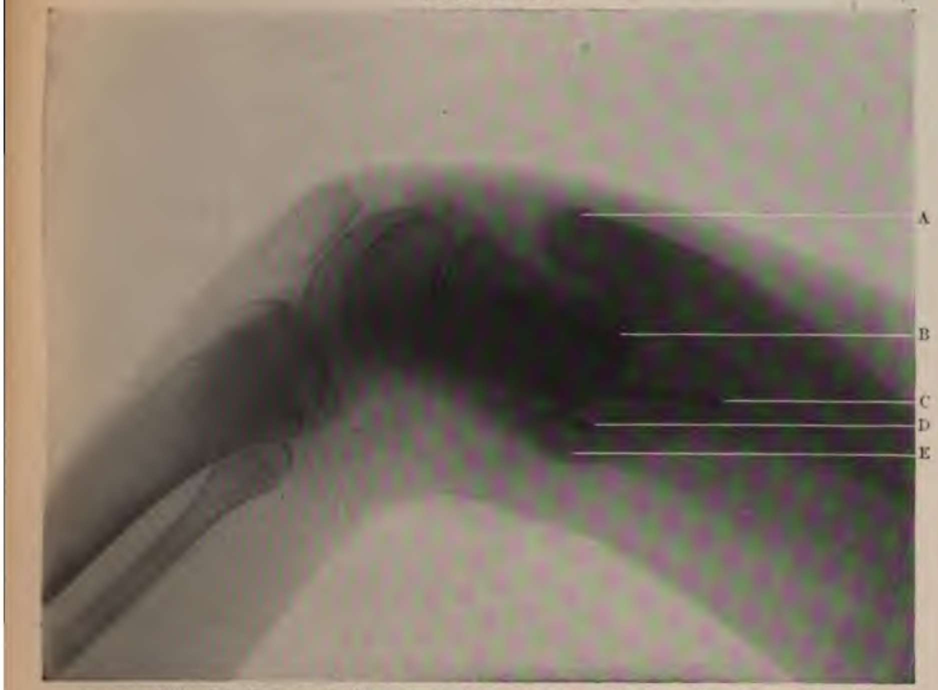
Symptoms.—The onset is sudden and sometimes marked by a chill. There are severe constitutional symptoms of fever and intense pain usually sharply localized. The part affected is swollen, reddened, and tender, and if a joint itself is involved, the swelling which is at first tense soon becomes fluctuating. From the beginning of the disease the signs of sepsis are manifest by the high intermittent fever, rapid pulse, pronounced leucocytosis, and great prostration. In exceptional cases these severe constitutional symptoms may not be present. In rapid cases death may occur in three or four days, but the disease is generally prolonged for one or two weeks.

Diagnosis.—The differential diagnosis is to be made chiefly from articular rheumatism, from which it is to be distinguished by the surrounding soft parts being much less involved and the general symptoms much more severe in osteomyelitis, and, if the disease occurs in the first two years, by the rarity of rheumatism at so early a period.

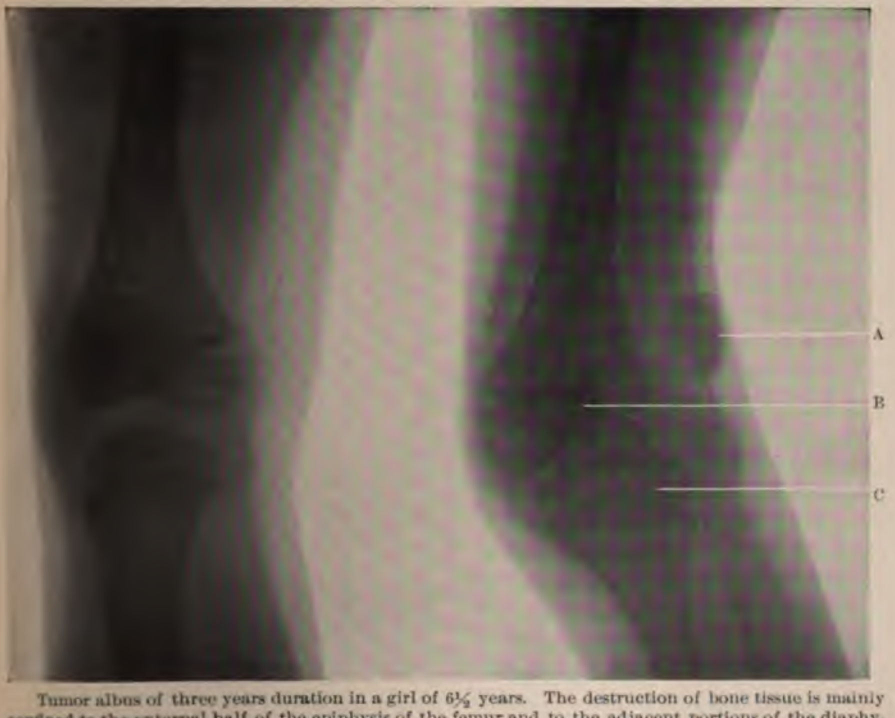
From tuberculosis, osteomyelitis is to be differentiated by the slow onset of the former and the primary lesion generally showing itself in the shaft of the bone in the latter, while in the former the epiphysis is usually first attacked. The process of osteomyelitis may, however, attack the epiphysis and extend to the joint. The presence of a leucocytosis in osteomyelitis is also of great aid in the diagnosis.

Prognosis.—The prognosis is very unfavorable unless immediate and radical surgical interference is carried out.

PLATE VII.



Osteomyelitis of the lower end of the femur, with spontaneous fracture. Boy, 9 years old. A, lower end of upper fragment; B, upper end of lower fragment; C and D, portions of cortical bone which have been sequestrated, and were subsequently removed by operation; E, callous tissue.



Tumor albus of three years duration in a girl of $6\frac{1}{2}$ years. The destruction of bone tissue is mainly confined to the external half of the epiphysis of the femur and to the adjacent portions of the diaphysis. A, subcutaneous cheesy abscess, communicating with the diseased area of the bone; B, probably a sequestrum surrounded by necrotic tissue; C, shows enlargement and squaring of epiphysis of tibia, as compared with the normal, indicating congestion and proliferation before absorption of the bone tissue has begun.

TREATMENT.—The treatment is essentially surgical. Where osteomyelitis of the shaft of a long bone is found at operation, all of the affected portions should be removed, even if a considerable portion of the shaft is taken, experience having shown that new bone will soon form, and that a serviceable limb is the usual result in the leg and forearm.

Prevention of septic infection of the umbilical cord, and of the wound after the cord has fallen off, is important as a matter of prophylaxis.

The following case came under my observation at the Infants' Hospital:

An infant, one month old, was admitted to the hospital November 16. The parents, who were Italians, were seemingly healthy and had one other child, a girl, who was also reported to be healthy. One week previous to entrance, what was described as a "red rash" appeared on the abdomen and spread down the left leg. It lasted about four days. Two nights previous to entering the hospital the infant had cried seemingly from pain on movement of the legs. There had been some discharge of pus from the umbilicus which had never healed. The infant appeared to be well nourished and of good color, with no abnormal appearances, excepting that the umbilicus had not healed, and also that the lower third of the left thigh was swollen so as to be twice as large as the right thigh, and that the skin in this region was red and shiny. The left leg was held stiffly and somewhat flexed, the knee being bent almost at a right angle, and both in front and over the anterior tibial group of muscles and over the calf there was a brawny feeling without fluctuation or localized tenderness.

On the day following entrance, the brawny infiltration was less extensive. The temperature, however, remained over 40° C. (104° F.). On November 19, a vertical incision was made from the inner side of the head of the tibia about four inches downward. This revealed a tibial abscess under the periosteum, which was separated for two inches both in front and behind. The cartilaginous epiphysis was separated and freely movable, and the bone of the shaft was red, rough, soft, and covered with small bleeding points. The upper two inches of the shaft were removed, revealing what appeared like hard, healthy bone. A counter-opening was made in the calf, and after irrigation a sterilized dressing was applied. The temperature continued between 39° and 40° C. (102.2° and 104° F.).

On changing the dressing in the afternoon, the lower surface of the upper epiphyseal cartilage looked gray and opaque, but at the next dressing several bleeding points were seen in the epiphysis and the wound looked well. A swelling had suddenly appeared over both malleoli, and an incision let out some greenish pus. A small area of the bone was curetted at this time.

Some hours later a swelling appeared over each wrist, which was drained by two incisions, one over each styloid process. No communication was found with the bare bone. The skin was found to pit on pressure, and to be slightly reddened over the biceps.

On the following day, November 22, the infant was found to be jaundiced. Fluctuation appeared and the arm was freely opened from the coracoid process to the elbow. An opening was also made at the posterior border of the deltoid, as pus was discovered along the long head of the biceps. The shoulder was then irrigated and drained.

The cultures which were taken from each of these abscesses showed no growth excepting the one from the tibia, where staphylococcus aureus was found in abundance. It was afterwards noted that this growth had not been found elsewhere because the oven where the cultures had been baked had been overheated.

On the following morning the hand and arm were blue, cold, and swollen. No pulse could be felt, and the tissues in the wound were almost black. The child died nine days after the first symptom pointing to an affection of the bone.

The autopsy was made three hours after death, and was interesting in that it showed no abscesses which could not be traced to multiple osteomyelitis. There was almost entire absence of pathological changes in the viscera. A separation of the epiphysis had occurred at the lower end of the left tibia and fibula, the upper end of the left femur, the lower end of both radii and ulnæ, and the upper end of the right humerus, with softening of the bone and pus. No thrombi were found in the right axillary vessels, the whole right arm to the wrist being swollen and discolored, and on cutting into it a red serous fluid flowed out of this phlegmonous inflammation. Cultures from the heart's blood, kidneys, and liver showed no growth, but from the hip-joint and tibial marrow there was an abundance of pure staphylococcus aureus, and from the phlegmonous arm a faint growth of streptococci. Blood-serum was the culture medium.

The case showed a rapid septic infection confined to the bone-marrow and bone of the shafts. Several long bones were affected, unaccompanied by any inflammation of the lymphatic glands or of the internal organs.

Pure cultures of staphylococcus aureus were obtained from the left tibia and hip, and as there was no discernible difference in the appearance of the three bones, it is probable that the same micro-organisms had existed in all the inflamed bones. The streptococcus infection of the arm was probably due to accidental inoculation at the time of the operation. The shoulder- and hip-joints contained pus, but the knee and wrist escaped. This is to be explained by the anatomical relation of the epiphysis to the capsule of the joint. When the epiphyseal line reaches within the joint-capsule pus can reach the joint whenever the epiphysis is separated by suppuration, and this relationship existed in the hip- and shoulder-joints. In the wrist the capsule is inserted into the epiphysis of the radius and ulna, and not into the shaft, so that pus to reach the joint has to perforate the cartilaginous epiphysis. This is more difficult than when the suppuration can pass directly from the epiphysis into the joint. The same is true of the upper epiphysis of the tibia, for the capsular ligament of the knee does not cross the epiphysis but is inserted into it, therefore, when separation occurs, the pus burrows under the periosteum, but does not break into the joint.

RHEUMATIC FEVER.

Rheumatic fever (acute articular rheumatism) is an acute infectious disease, non-contagious, affecting usually a number of joints, and showing a tendency to inflammation of the endocardium and pericardium.

Etiology.—The cause is unknown, but there is such an intimate relation between articular rheumatism and endocarditis, a disease which has been proved to be of bacterial origin, that the disease is now considered to be produced by some specific organism. Tonsillitis is such a frequent initial symptom that it is possible that it may be the point of entrance of the infection. Although acute articular rheumatism may occur at any age, it is very rare in infancy, and comparatively rare in early childhood.

The subacute attacks, characterized by a moderate heightening of the temperature and milder symptoms, may occur in children, and are more apt to be complicated by endocarditis and pericarditis in childhood than in adult life. They may last for many weeks and finally become chronic. Exposure to dampness, to cold, and to sudden and great changes in temperature are important predisposing causes. One attack predisposes to another.

The symptoms are so varied and so similar to those of a septic infection, as evidenced by the character of the fever, the involvement of the serous membranes, the tendency to relapse, the sweats, and the leucocytosis, that it corresponds closely to the type of an acute infection.

PATHOLOGY.—There are no lesions which especially characterize the pathology of the disease. According to Osler, the affected joints show hyperæmia and swelling of the synovial membranes and of the ligamentous tissues. There may be a slight erosion of the cartilage. The fluid in the joint is turbid, albuminous in character, and contains leucocytes and a few fibrin flakes. An extensive effusion into the joint is, however, uncommon. Pus is very rare in uncomplicated cases. The blood usually contains an excessive amount of fibrin. In the secondary rheumatic inflammations, such as pleurisy and pericarditis, various pus organisms have been found, possibly the result of a mixed infection.

SYMPTOMS.—The onset of the disease is usually acute, and is characterized by loss of appetite, fever, swelling, tenderness, and redness of one or more joints. Pronounced redness and swelling of the joints are not so marked as in adults. The temperature and pulse are usually moderate, although in the beginning of the attack, following the rule of acute infectious diseases, a sudden rise of temperature to 39.4° to 40° C. (103° to 104° F.) is not uncommon. The pulse and temperature soon fall several degrees in the course of the disease, and then gradually become normal, unless some complication arises.

As the disease progresses the tongue becomes coated, although not especially dry. The urine is diminished, high-colored, and very acid. On cooling it deposits urates. The chlorides are diminished. A characteristic symptom is profuse and acid sweating, having a sour odor. Later in the disease, if the sweating is persistent, the perspiration, according to Osler, may become neutral or alkaline. The mind is usually clear. The joints are apt to be invaded successively, the joint first affected sometimes becoming decidedly less painful and swollen as the next joint is involved.

The child presents a picture of dread, fearing that the attendants will touch or try to move it; in fact, any change in the posture necessitating motion of the affected joints is strenuously objected to. Anæmia develops with great rapidity, and a marked leucocytosis is present. The disease runs a varying course of from three to six weeks, unless complicated by some other disease. In some cases an endocarditis may appear before the development of the rheumatic symptoms. Delirium occurs not infrequently in cases of rheumatic fever, and may often be traced to the toxic effects of overdoses of the salicylates.

COMPLICATIONS.—The most frequent complication of articular rheumatism in children is *endocarditis*. The onset of the complication is usually characterized by precordial pain, rise of temperature, and, on physical examination, a soft-blowing systolic murmur of mitral origin. Although hæmic murmurs may develop in the course of acute articular rheumatism, yet the organic murmur of endocarditis is so much more common that its presence should be looked upon as a possible grave lesion in every case, and the heart should be carefully examined at each visit for the purpose of detecting a murmur as soon as it is perceptible. In some cases the endocarditis begins very insidiously without noticeable symptoms referable to the heart, and in children there is a special tendency to dilatation.

Next to endocarditis the most frequent complication is *pericarditis*, which begins with a pericardial friction-rub, usually under the middle or upper part of the sternum, with pain and precordial distress, and later with the usual mechanical symptoms of dyspnœa and orthopnœa, as described under pericarditis. The disease is often associated with endocarditis, but does not usually occur at so early an age as endocarditis, the middle period of childhood being most prone to the disease. Especial mention should be made of the *delirium* connected with pericarditis. When a pericarditis is accompanied by effusion, the fluid, in children, is especially likely to be purulent. Of the other more rare complications can be mentioned *myocarditis*, *pneumonia*, *pleurisy*, and *peritonitis*, also certain cerebral complications usually associated with hyperpyrexia and represented by delirium, coma, and convulsions. The relation between *chorea* and rheumatism is stated on page 917.

Various forms of *erythema* appear in the course of rheumatism. A rather uncommon symptom in this country, although seemingly more frequently met with in England, is the development of small subcutaneous *fibrous tumors* attached to the tendons and fasciæ in any part of the body or limbs. They seem to be closely associated with rheumatism, are especially frequent in children, and are connected with rheumatic endocarditis.

DIAGNOSIS.—The diagnosis of rheumatic fever is made by the presence of the characteristic symptoms of high fever of acute onset, followed by localizing symptoms in one or several joints of redness, swelling, tenderness, marked pain on motion, and leucocytosis. The disease is to be differentiated from several affections which closely resemble it.

From Acute Osteomyelitis.—The diagnosis is made principally by the greater intensity of the localizing symptoms, the involvement of the epiphyses or the shaft of the bone, rather than the joint, and the presence of more pronounced constitutional symptoms in osteomyelitis. Moreover, there is less tendency to a multiple affection of the joints.

From Multiple Secondary Arthritis.—Infections of the joints may occur in connection with gonorrhœa, scarlet fever, dysentery, and cerebro-spinal fever, but the differential diagnosis can usually be made without difficulty by the course of the symptoms, the tendency to involve but one joint, and the recognition of the disease in which the arthritis occurs as a complication.

PROGNOSIS.—The prognosis of rheumatism in children is very favorable, unless complications arise, in which case it depends upon the severity of the complication. Endocarditis and pericarditis make the prognosis especially grave, as rheumatic attacks are liable to recur, and at each recurrence the cardiac lesions show a tendency to become more pronounced and serious.

TREATMENT.—The child should be kept in bed, and made to wear soft flannel night-gowns so fashioned that they can be changed with as little disturbance as possible, and fresh gowns should be put on as often as severe sweats occur. It is desirable that patients should sleep between blankets in preference to sheets. Milk is beyond doubt the most appropriate diet. The thirst can be satisfied by lemonade, or oatmeal- or barley-water. Soups and broths may be used to vary the milk-diet.

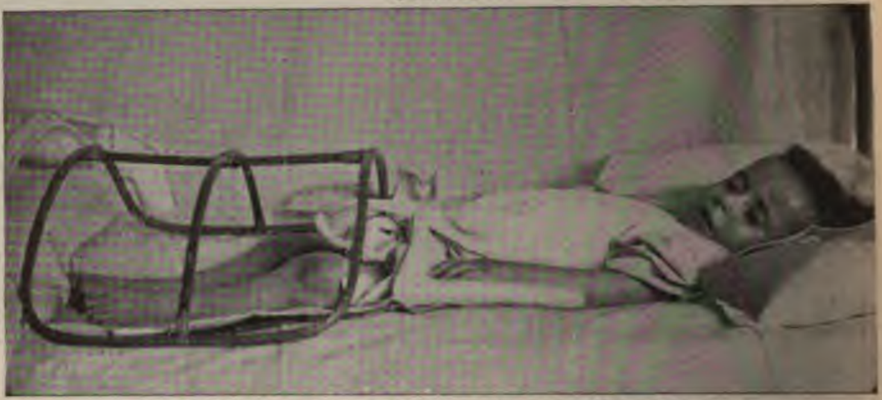
There are no drugs which act directly upon the disease, either to shorten its course or prevent its serious complications. For the relief of pain, however, the salicylates are especially valuable. They should be given in fairly large doses, 0.3 gramme (5 grains), for the first twenty-four hours until toxic symptoms, such as slight disturbance in hearing and ringing in the ears, are produced, or until the pain has subsided. When the pain is once under control, small doses of the salicylates three or four times a day will usually keep the patient comfortable. With the fresh involvement of another joint, an increase in the quantity of the salicylates may be needed temporarily. The ammonium salicylate is perhaps the most useful preparation. Salol, salicin, and oil of wintergreen (two to four minims) may be tried. The alkaline treatment is often effective, and may advantageously be combined with the salicylates. Ten or fifteen grains of the citrate of potassium in two or three ounces of water, and the addition of thirty grains of bicarbonate of soda with a half-ounce of lemon-juice, makes an agreeable effervescent drink. This may be given every three or four hours until the urine is slightly alkaline, and then in such quantities as to keep the reaction just perceptibly alkaline.

Phenacetine in small doses, 0.06 to 0.12 gramme (1 to 2 grains), guarded with stimulants, may be used to control exacerbations of pain when it is not desirable to increase the salicylates. Especial attention should be directed to the heart, and its complications treated symptomatically.

The local treatment of the joints, in the acute stage, consists in the application of hot laudanum fomentations for the relief of pain. The weight of the bedclothes should be kept off the affected joints by means of a cradle. When the pain has subsided, the joints should be wrapped in cotton-wool. When the process has become subacute or chronic, great improvement, as evidenced by diminution in the swelling, stiffness, and pain on motion, often follows the application of dry heat for an hour every day or several times a week. This may best be applied by the ovens especially adapted for the purpose.

The disease will generally run a course of from four to six weeks, during which time the patient should generally be kept in bed, and care be taken to maintain an equable temperature in the room. Owing to the tendency to a recurrence of the disease, it is often advisable to remove the child to a warm, dry climate during the cold and unsettled weather which prevails in the winter and early spring in the North.

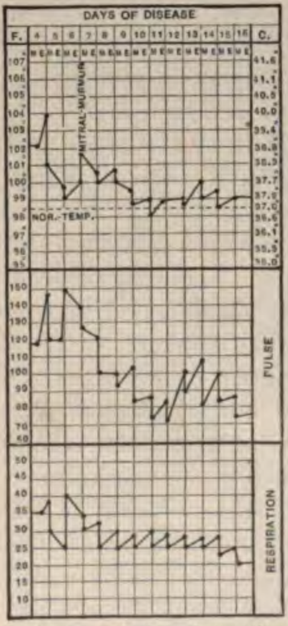
FIG. 118



Acute articular rheumatism. Adult type of disease. Male, 3⅓ years old.

The following cases illustrate rheumatic fever:

CHART 19.



Acute articular rheumatism. Acute endocarditis on seventh day from beginning of attack.

A boy, three years and four months old, was treated in the hospital for bronchitis, and when convalescent from that disease was attacked with acute articular rheumatism.

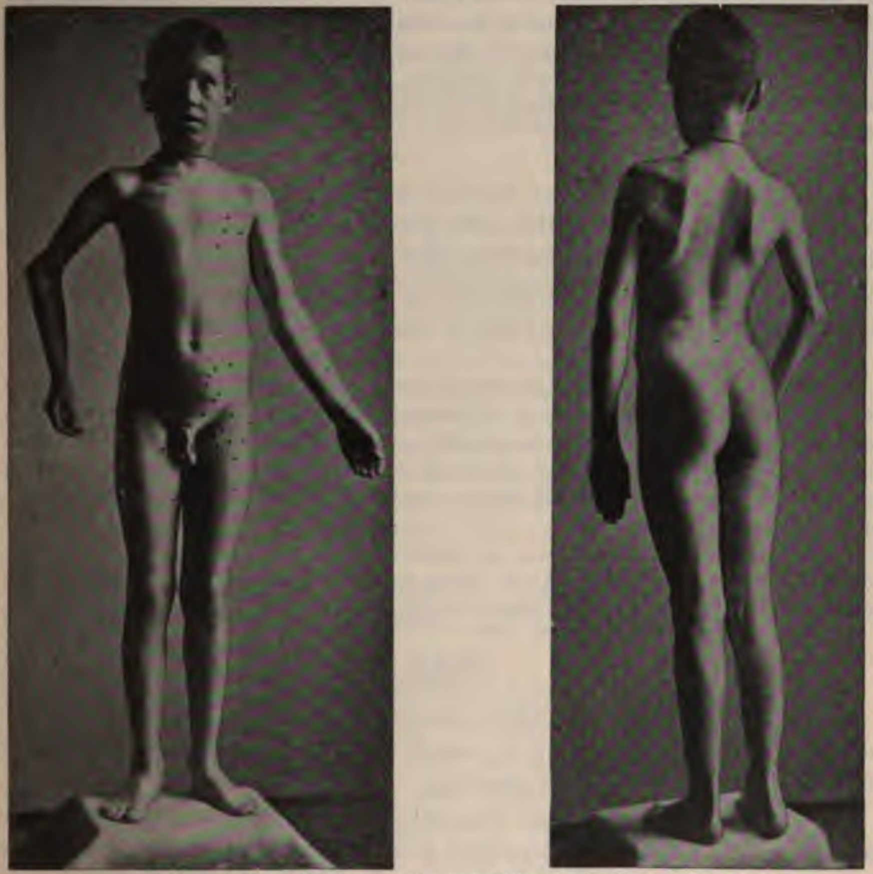
There was no rheumatic history in his family, and he had never had rheumatism nor any other disease except bronchitis. After having been feverish for two days, the temperature varying from 37.7° to 38.8° C. (100° to 102° F.), he complained of pain and tenderness in his shoulders, wrists, and elbows. On the following day these symptoms increased, being especially marked in the left hand and left knee. There was an expression of anxiety on his face, showing that he feared that the tender joints would be touched. The weight of the bedclothes was kept from the knee by a cradle, and the arm was comfortably arranged on a pillow. These details in the nursing of a rheumatic child are very important. Fig. 118 shows the affected knees and wrist. He was being treated with oil of gaultherium, 4 minims every three hours. The temperature varied from 38.3° to 39.4° C. (101° to 103° F.). An examination of the cardiac region did not reveal any cardiac complication.

The child suffered considerably for four weeks, but at the end of that time the joints gradually grew less painful, and he was entirely well thirty-three days from the onset of the attack.

The next child, a girl, five and one-half years old, was interesting as illustrating a number of characteristics in connection with the rheumatism of children.

She was attacked eight days before entering the hospital with pain, swelling, and tenderness in her left ankle. On entrance her temperature was 40° C. (104° F.), her pulse was 145, and her respirations were 40. There was very slight pain in the joints, and, although her appetite was lessened, she otherwise seemed well, and had not complained of any pain since the beginning of the attack. On the sixth day the temperature fell to 37.2° C. (99° F.). It is interesting to note the extreme latency of the disease, and how the child seemed to be perfectly comfortable from the beginning of the attack, except when the ankles, both of which were swollen and tender, were touched. On the seventh day of the disease the temperature rose to 38.6° C. (101.5° F.), and an examination of the chest showed a mitral systolic murmur. The murmur became more marked, and was transmitted into the axilla and the back. The area of superficial cardiac dulness was slightly increased, and extended to the middle of the sternum.

FIG. 119.



Rheumatism. Subcutaneous fibrous nodules. Male, 13 years old.

By the end of the second week of the attack the pain and tenderness had left the ankles, and the child seemed quite well. The area of superficial cardiac dulness was found to be normal, but the systolic murmur still continued. Chart 19 shows the rheumatism gradually subsiding up to the seventh day of the disease, when the endocarditis arose as a complication.

Fig. 119 represents a boy who during an attack of rheumatism developed the subcutaneous fibrous nodules which have been referred to.

When he was seven years old he had an attack of rheumatism affecting his ankles and the muscles of his neck. His temperature was 37.2° C. (99° F.); his urine was normal. During this attack a systolic souffle transmitted into the axilla developed, and the area of superficial cardiac dulness was increased. This attack lasted eight days. From that time until he was thirteen years old, that is for six years following his first attack of rheumatic fever, he had more or less dyspnœa on exertion, and at times cardiac pain, but never had any marked return of the rheumatism. At the end of that time he noticed small lumps appearing under his skin. When they were first observed he had indefinite pains in his limbs, severe headache, and malaise. Some of the lumps were slightly tender, and were distributed on the chest, arms, abdomen, and legs, mostly on the anterior surface.

Although acute articular rheumatism is rare in infancy, I have met with a number of cases at this early period of life. Reference has already been made, on page 95, to the little girl, two years old, who, after exposure, was attacked with acute rheumatism in both hip-joints. Two other cases are of special interest on account of their early occurrence.

The first case was one of rheumatic fever, attacking all the joints, in an infant two weeks old, after exposure to a cold draught while being bathed. Any movement of the joints caused the infant to scream. He lost rapidly in weight, his surface circulation was disturbed, and the attack lasted for four months; but when he was six months old he was perfectly well, and no cardiac complication developed during the attack.

Another case of this kind was an infant who was attacked with rheumatic fever when she was seven months old, the attack lasting until she was fifteen months old, when she recovered without any cardiac complication, and became well and strong.

SYPHILIS.

The specific organism which causes syphilis has not yet been discovered. The disease as it is manifested in early life appears in two forms,—(1) *acquired* and (2) *hereditary*.

The former differs in no respect from the disease as it occurs in adults, and is transmitted by direct infection, usually through one of the mucous membranes. Its treatment and general characteristics are the same as in adults.

HEREDITARY SYPHILIS.

The hereditary form of syphilis plays an important part in the diseases of the early months of life, and is an affection which in all its phases should be thoroughly understood by those who practise among children.

By inherited syphilis we mean a congenital disease which has been transmitted to the child through one of the parents or through both. It makes its appearance either in the early months of life, *syphilis of the* *new-born*, or at a later period towards puberty, *retarded syphilis.* The stage which is met with at birth usually corresponds to an early stage of acquired syphilis, while that which is delayed until later childhood or puberty corresponds to a later stage.

Inheritance and Transmission.—The question whether the infant can inherit syphilis from the father without the infection of the mother is one which has not yet been determined finally. The weight of evidence is in favor of the view that its occurrence in this way is not possible. The probability is that some mild and transient form of the disease has been overlooked in cases where the mother has been apparently healthy, especially as the mother of a syphilitic infant is always immune to infection by her infant. Instances, however, occur in which it is impossible to say that the mother of an undoubtedly syphilitic infant is also syphilitic. The following case illustrates this point:

An infant with marked syphilitic lesions was brought to me for treatment. The father of the infant acknowledged having been treated for a primary syphilitic lesion which was followed by pronounced secondary symptoms. The mother was a healthy, strong woman, who had always been perfectly willing to give any information required either as to her own or as to her husband's condition, in order to aid in the preservation of her infant's life. She stated that she had never had any miscarriages, that she was perfectly well both before and after the birth of this infant, and that she had never had an efflorescence on her skin, a sore throat, nor any lesions of the mucous membranes. She came under my observation when her infant was six weeks old, and has since then been seen sufficiently often for me to say that so far as I can determine she has had no symptoms that in any way could be attributed to syphilis. She had always had a plentiful supply of breast-milk, which was evidently of good quality.

A syphilitic infant does not infect its mother (*Colles's law*). It can infect a woman, however, who either has never had syphilis or who has never given birth to a syphilitic infant.

It is probably possible for a syphilitic fœtus to infect its mother in utero. This theory of *retro-infection*, however, has not been universally accepted. Fournier believes that there is a class of cases in which the father at the time of marriage has no lesion which would necessarily infect the mother, where the mother never shows any initial lesion and remains free from syphilis so long as she is unimpregnated, and where after impregnation she becomes syphilitic and either aborts or gives birth to a syphilitic infant. In connection with the subject of retro-infection the question arises whether a mother who becomes syphilitic during her pregnancy can infect the fœtus (*post-conceptional syphilis*). There is no doubt that she may abort from her own syphilitic infection. The same authority believes that the fœtus is also syphilitic. This doctrine, however, is not universally accepted. There is no doubt but that infection may take place; whether it always does is not yet settled.

It has been found that when a woman is syphilitic it is exceedingly common for her to abort. Miscarriage is more frequent when a woman is passing through the early stages of syphilis than later when she has become more or less habituated to the disease. The treatment by mercury in these cases soon after impregnation, and continued during the pregnancy, is a valuable means of averting abortion. Although the aborted fœtus of a syphilitic woman is usually macerated, yet such a condition of the fœtus may be produced by other diseases as well as by syphilis. Birch-Hirschfeld has found from an examination of a large number of macerated fœtuses that seventy per cent. were undoubtedly syphilitic.

Although the tendency to transmit the disease is greatly lessened by time, yet the thorough treatment of the parents by mercury is the most efficient means of preventing such transmission, and the careful use of this drug in proper doses is never contraindicated. It is, therefore, evident that a pregnant syphilitic woman should be treated with mercury whether she was infected before or after conception. When both parents are syphilitic, and when their syphilis is in the early stages, the infant is most likely to inherit the disease, and under like conditions the disease is apt to be of a severe type.

Infants entirely free from syphilis, either at birth or later, have been known to be born of parents of whom one or both were undoubtedly syphilitic. The following cases illustrate this question of immunity:

Two children of a family of five were under my care, all of whom were healthy at birth and had never shown any symptoms of syphilis. The father was infected with syphilis before marriage, and later infected his wife. They were both carefully treated with mercury. The wife had never had any abortions. She had had five children, and had lost none. Both father and mother had had undoubted secondary and tertiary lesions, some of which still existed at the time I saw them.

The father of another such case was a rag-sorter, who had a primary syphilitic lesion on his hand twelve years previously. This lesion was followed by secondary symptoms. He had never had any lesion on the penis. While he was being treated his wife showed symptoms of syphilis and was also treated with mercury. This child had always been healthy, and was one of three, none of whom had ever developed any syphilitic lesions.

Pathology.—The pathological tissue-changes which take place in the hereditary form of syphilis are of the same nature as those which occur in the acquired form. Diffuse *interstitial hyperplasia* is much more common in the hereditary form than are circumscribed gummy tumors. Changes in the bones are very common in hereditary syphilis, and in fact so much so that it is usually considered necessary to find these osseous changes in order to establish a diagnosis of syphilis in the fœtus.

Osseous System.—The changes in the bones which take place in hereditary syphilis are so important, not only on account of their pathological interest, but also because of their clinical significance, that especial attention should be paid to them.

In this connection it should be remembered that in the latter part of intra-uterine life the long bones are cartilaginous and the process of ossification is intra-cartilaginous. As the cartilage changes to bone the cartilage-cells increase in number and are closely crowded together. Then comes the area of osteoblasts, then the calcareous matter, and deeper down in the ossified portions are the blood-vessels running in from the periosteum. The epiphyses of the bones of the arm are cartilaginous at birth, and they remain separated from the shaft of the bone for some time by a narrow cartilaginous layer. It is in this cartilaginous separating layer, called the zone of proliferation, represented in the drawing of a normal infant's bone (page 329, Fig. 82 I.), that certain changes are found in hereditary syphilis. It is also at this zone of proliferation that the growth in the length of the bone takes place, and it is here that syphilitic changes are most often found. This lesion is an osteochondritis, and may occur together with lesions of the spleen and other parts of the body, or as the only manifestation of the disease.

Osteochondritis is ordinarily the form of bone-disease in infants. Osteoperiostitis belongs almost exclusively to the later forms of hereditary syphilis as they appear in older children and in young adults.

The bones which are affected most commonly are those of the arms and of the legs.

Besides these common osseous lesions a morbid condition of the fingers and toes, called *dactylitis*, occurs quite frequently. In this condition the fingers and toes assume a peculiar pyriform shape.

In addition to these purely syphilitic changes, local thinning of the bones of the skull, called *craniotabes*, occasionally occurs. In this condition the bone-substance is absorbed, leaving only the integuments and membranes.

Liver.—The liver is always larger than in the normal condition. The hepatic tissue is harder and more elastic than usual; it is of a yellow color, and there are small white granulations scattered throughout the parenchyma. The hepatic acini under normal conditions are in contact, except at the prismatic spaces formed by their union, in which spaces the capsule of Glisson forms an envelope to the afferent portal vessels of the lobule. It is in these spaces that the round lymph-cells form and collect into small nodules representing microscopic gummata. The gummata of the liver which are found in young children with hereditary syphilis resemble those which occur in adults.

Spleen.—Next to the osseous system the spleen is the part most often affected by syphilis. It is enlarged, and the degree of splenic enlargement is usually characteristic of the severity of the disease.

Pancreas.—Birch-Hirschfeld has pointed out the fact that the pancreas is frequently found to be affected in hereditary syphilis. He remarks that the interstitial changes which he found in the pancreas are analogous to those which occur in other organs, especially the liver, and that, while these changes are not constant, they come next in frequency to the alterations in the spleen. The interference with the function of the pancreas, which must occur when it is diseased to any great extent, is probably the cause of the gastro-enteric disturbances so common in hereditary syphilis.

Lungs.—In cases of hereditary syphilis born before term, and in those born at term who live but a few days, the lungs present certain pathological conditions represented by nodules or small tumors, usually superficial and varying in size. Sometimes an entire lobe may be involved, and the dense, altered lung-tissue is colorless gray or white, both on its surface and on its section. This condition has been called by Virchow *pneumonia alba*, white hepatization.

Kidney and Testicle.—The kidney and testicle may show the lesions of syphilis. It is to be noted that the lesions of these organs are amenable to treatment. The disease in the testicle is represented by a gradual enlargement, and is usually bilateral.

Throat, Upper Air-Passages, Thymus Gland, and Heart.—Extensive lesions are at times found in connection with the pharynx, larynx, trachea, and neighboring parts, and also with the thymus gland and with the muscles of the heart.

Early Manifestations of Hereditary Syphilis.—SYMPTOMS.—The severity of the disease determines the type of the efflorescence, and is also influenced by the time when the infection of the fœtus took place. Thus, the later the period of infection the milder will be the form of the efflorescence which first appears, while the less severe the general symptoms the better will be the prognosis and the greater the amenity of the disease to treatment. The reverse of these rules is found when the infection has taken place early, and when, as a result, the infant is born dead, or at birth shows such advanced stages of the disease as are represented by the more intractable forms of efflorescence and severe general symptoms, making the prognosis exceedingly grave.

In the mild form of the disease the infant may be born apparently healthy and may show no indications of its syphilitic inheritance for some weeks. It is rare, however, for the symptoms to be delayed beyond the first three or four months of life. The earliest symptoms of hereditary syphilis correspond to the secondary symptoms of acquired syphilis. Commonly, unless the infant is born with the efflorescence, it is noticed at birth, or within two or three weeks, to have occlusion of the nares (*snuffles*), and, soon after, a hoarse cry and an efflorescence of a macular or a papular variety. The efflorescence is general, includes the palms of the hands and the soles of the feet, and is especially prominent on the forehead.

The condition of the infant depends considerably on that of the mother. The rule is that these infants when born are emaciated, but I have seen them well developed and apparently in good condition. The disease, with appropriate treatment and good feeding, may in some cases be arrested in this stage, and be cured so that it will not return, or it may advance to another group of symptoms, which are represented by lesions of the mucous membranes, and sometimes by pseudo-paralysis of one or both limbs of a greater or less degree. All these symptoms may arise, run their course, and completely disappear, sometimes never to return. Again, they may reappear at various times during the individual's life, but they are especially liable to recur during the middle period of childhood and at puberty.

The course of syphilis is so influenced by treatment that the symptoms must necessarily be irregular. When the disease is untreated, as a rule, all the symptoms grow worse. The infant becomes more and more emaciated, and either it dies in a few weeks of inanition, or the disease progresses still further and serious lesions of the various organs, such as the lung, liver, spleen, and kidney, may finally produce a fatal result.

Efflorescence.—We can judge to a great degree as to the severity of the disease by the type of the efflorescence, and also by the time when it occurs after birth. The mildest and most benign form of syphilitic efflorescence is represented by *maculæ*, the next by *papulæ*, and the next by *pustulæ* and *bullæ*. Another form of efflorescence simulating *psoriasis* is one of the more severe manifestations of syphilis, as is also that form which is called *rupia*, where the efflorescence consists of thick layers of crusts arranged one above the other, forming a conical mass, the skin at the base being somewhat infiltrated. All of these types of the disease have been known to be cured. Finally, one will meet at times with a very dangerous form of the disease, which is almost uniformly fatal no matter what the treatment may be. This is what is called *syphilitic pemphigus*, and is represented by large and numerous bullæ. The syphilitic efflorescences, unlike most other lesions of the skin, appear commonly on the palms of the hands and the soles of the feet.

Alopecia.—In addition to these general symptoms there occurs in the hereditary form of syphilis the loss of hair which is so common in the acquired form of the disease. This alopecia may be caused by any of the dermal lesions which occur during the course of the disease, but is probably due mostly to the general lack of nutrition in which the skin participates with the other organs of the body in syphilis. In certain cases the eyebrows and eyelashes are lost, and Barlow believes that the former condition is characteristic of the disease, or at least should excite a suspicion of its presence.

Lymph-Nodes.—Enlargement of the lymph-nodes, *adenopathy*, seems to be less marked in hereditary syphilis than in the acquired form. This enlargement may be due to reflex irritation from the more severe dermal lesions, but in certain cases it is found where no dermal lesion exists. The enlarged nodes may be in the inguinal, the axillary, or the cervico-maxillary regions. They are distinct, movable, multiple, and non-inflammatory. The older the child the more likely the glands are to be enlarged.

Nails.—According to Post, the nails are involved quite frequently in hereditary syphilis, and more frequently than in the syphilis of the adult. The *onychia* occurs in two forms. In the first form a papule or pustule appears on the skin at the side of the nail. This ulcerates and extends along the side of the nail, at times involving the matrix and causing the loss of the nail. The thick and everted edges of the ulcer, its sloughing base and sanious discharge, are somewhat characteristic, and are accompanied by a painful enlargement of the distal phalanx. (For another form of onychia, see page 538.)

Teeth.—The effect of hereditary syphilis on dentition is quite marked. The first teeth instead of being cut in the sixth or seventh month may not appear until the fourteenth or fifteenth month, and sometimes even later. These primary teeth are especially liable to decay early. There is nothing sufficiently characteristic to be of diagnostic value in the appearance of the teeth of the first dentition.

Eye.—Hutchinson has observed twenty-three cases of *iritis* in syphilitic infants. The average age for the beginning of the iritis was five and a half months. The oldest was sixteen months at the time of the outbreak, the youngest six months. Both eyes were affected in eleven cases, and in fifteen cases the effusion of lymph was copious. The cornea was affected in a few cases. In seven cases the cure was complete, in twelve the pupil was partially occluded. Iritis is one of the rarest of the symptoms of hereditary syphilis, and at times escapes notice on account of the very slight symptoms which usually attend it. The diagnosis in these cases is not dependent on the iritis alone, but the infants always show other well-marked symptoms of syphilis. There is great danger of the disease resulting in blindness if it is left untreated, and mercurial treatment is most efficient in effecting a cure.

Digestive Organs.—In regard to the digestive disturbances which arise in these cases of hereditary syphilis, it is well to remember that they may depend upon a syphilitic lesion of the liver, spleen, and pancreas, as well as of the stomach and intestines. It is, therefore, necessary to treat these disturbances of the gastro-enteric tract in a different manner from what is customary where a local non-syphilitic cause is supposed to be present. In fact, mercurial treatment will produce the best results in these cases.

Hemorrhages.—An affection called *syphilis hæmorrhagica neonatorum* is met with at times. Bumstead and Taylor have reported two cases of this kind, and state that the disease is rare, less than twenty cases having been noted. The hemorrhages vary in their extent, and may occur in either the skin or the mucous membranes. This class of cases is difficult to differentiate from the hemorrhagic disease of the new-born which has already been described. There is no doubt that syphilis has in a number of cases an etiological significance in the umbilical hemorrhage which occurs in the early days of life. Dr. Uracek has reported a series of hemorrhages in the different internal organs apparently depending upon a syphilitic taint in the infant.

Nose.—The occlusion of the nares may increase to such a degree that the breathing of the infant is seriously interfered with, and, without any other syphilitic lesion, it may die from imperfect oxygenation of the air which enters its lungs.

This occlusion of the nares may cause great loss of sleep. We must, however, understand that, even where this lesion is not of any great extent, syphilitic infants suffer from insomnia. This insomnia is usually accompanied by crying, so that it is probable that the restlessness and insomnia are due to pain in the bones, as these symptoms are often present where there is no digestive disturbance. In connection with these syphilitic lesions of the nose, flattening of the bridge of the nose is at times a noticeable symptom.

Anus.—There is nothing especial to describe concerning the condylomata which are found in the anal region and which are rare in comparison with the lesions of the mouth. They begin as rounded papules, which sometimes coalesce, and there is more or less infiltration of their edges and breaking down of their centres.

Mouth.—The syphilitic lesions of the mouth are found so commonly, and are of so important a character, that an especial description should be given of them. There is no syphilitic lesion of the mouth which is represented by a characteristic stomatitis. The mucous membrane in the course of hereditary syphilis may at any time be in so sensitive a condition that the various forms of stomatitis may be engrafted on it, and we thus may have different lesions of the lips, tongue, buccal cavity, and tonsils, which, while simply representing the lesions of certain non-syphilitic affections, may, by their peculiar grouping in combination with other symptoms, represent the hereditary form of syphilis. The lesions most commonly appear around the lips and on the mucous membrane lining the cheeks. On the lips fissures are exceedingly frequent; on the upper lip they commonly appear on either side of the median lobule, while on the lower lip they are usually single and in the median line. The angle of the mouth is often the seat of condylomata, and these are frequently covered with crusts and at times are deeply ulcerated. A peculiar appearance is in some cases seen at the commissures of the mouth, caused by cutaneous ulcerations, which make it look larger than normal, and at times produce a number of lines radiating from the mouth to the cheeks. Ulcerations may occur on the tongue, the lips, and the fauces. Forchheimer considers that the fissures which occur in syphilitic infants' mouths when they are present, leave no doubt as to the diagnosis, since they are infiltrated. The most common place for them to appear is at the corner of the mouth. In this place, as a rule, the most striking feature of the fissure is that it is a papule which has been split in or about its middle, and that it has an infiltrated edge. The fissures sometimes disappear in the mucous membrane, sometimes stop before reaching it, and sometimes run into it. The fissures may or may not be covered by a crust, and, unlike most syphilitic efflorescences, produce more or less pain when the mouth is opened. These fissures are called *rhagades*. They are characterized by their persistency and by their lack of tendency to spontaneous healing. Ulcers and *plaques muqueuses* may be found upon the mucous membrane of the lips and cheeks and on the sides and under surface of the tongue. They are superficial, but cover more space than the fissures. The infiltration is not so well marked, but is present to a greater or less degree. The most common lesions which are found on the tongue are these plaques muqueuses and ulcers. Both have infiltrated edges, but the plaque in this situation rises above the level of the tongue, while the ulcerations are considerably depressed. They are both characteristic of syphilis. Their locality is determined somewhat by the presence of such irritants as sharp teeth pressing against a portion of the tongue. The secretion of all these lesions of the mouth and lips is highly infectious.

Bones.—One of the striking symptoms of this early stage of hereditary syphilis results from osteochondritis. According to Post, the form of lesion is usually that of a tumor at the junction of the diaphysis and epiphysis at the distal end of the long bones, although any part of the osseous system may be involved. These swellings are difficult to recognize in fat children. The tumors rise abruptly from the bones; they are small and globular, and in some cases form a ring at the junction of the shaft and epiphysis; in others the whole epiphysis is enlarged. At times only a part of the cartilage is affected, and the external swelling is correspondingly circumscribed. The lesions appear soon after birth, and their development is completed either slowly or rapidly. The termination varies widely. The swelling may be absorbed under appropriate treatment, or suppuration may take place and the skin break down; the disease may end in the separation and destruction of the epiphysis. The result upon the final growth of the bone varies, of course, with the severity of the local disease. When the morbid process is arrested before the destruction of either cartilage or epiphysis, there is no deformity, but the destruction of cartilage puts an end to growth at that point, and a more or less shortened and useless limb results. When the disease takes such a course as to separate the epiphysis while the integuments remain sound, the limb becomes useless for a time and appears to be paralyzed. The disease was first fully described by Parrot, and is known as Parrot's disease, or *syphilitic pseudo-paralysis of the new-born*. The joints in immediate connection with the diseased bones are sometimes involved. There may be simply an effusion, but, where the bone is destroyed, serious disorganization of the joint must follow. The pain and sensitiveness in these cases of pseudo-paralysis are probably caused by a low grade of periostitis.

The bones of the fingers and of the toes present at times the peculiar lesion which is known as *dactylitis syphilitica*. The phalanx may be enlarged to two or three times its natural size, giving the fingers a pyriform shape. One or several fingers or toes may be involved, and sometimes the metacarpal bones are diseased. The proximal phalanx is more frequently affected than the distal phalanx. In the early stages the integument is unchanged; later, the overlying parts become involved and abscesses form. If the case is submitted to early treatment the deformity usually subsides, but if untreated the disease may result in permanent deformity and uselessness. Dactylitis, however, is not characteristic of syphilis alone, as it occurs also as a result of tubercular disease of the bone.

One of the more uncommon symptoms of hereditary syphilis is *craniotabes*, but in rare cases it may be found. These softened spots, nearly circular in form and about 1.2 cm. (½ inch), more or less, in diameter, may be recognized by the finger during life. Formerly craniotabes was considered to be exclusively a symptom of rhachitis. It is found especially in the occiput. It is present in rhachitis where no trace of syphilis can be discovered, but it seems to be most common in cases in which there is a distinct syphilitic taint. Out of one hundred cases of craniotabes collected by Drs. Barlow and Lees, in forty-seven there was satisfactory proof of syphilis.

DIAGNOSIS.—The diagnosis of hereditary syphilis in its more advanced forms, such as has just been described, is not difficult, as no other disease represents such serious lesions of the skin with such a combination of general symptoms and lesions of the mucous membranes.

The milder forms of the disease are frequently mistaken for other diseases of the skin, such as papular erythema, which simulate the syphilitic lesions but which are of a benign character.

Occlusion of the nares caused by swelling of the Schneiderian membrane, if persistent during the early weeks and months of life without rise of temperature, should always make us suspicious of the presence of hereditary syphilis, for a syphilitic efflorescence is often so slight and evanescent as to be frequently overlooked.

Marked improvement following the administration of mercury is also usually considered of diagnostic value, and, although not by any means conclusive, is at least significant.

Periostitis, especially of the lower end of the humerus or the anterior border of the tibia, is met with in children. It should make us suspicious that syphilis is causing this condition, especially if there is periostitis of a number of bones at once.

A great deal has been written and much discussion has taken place regarding the relationship between syphilis and rhachitis. The two diseases are so distinctly separated that it seems scarcely necessary to dwell, except very briefly, on the differential diagnosis between them. Rhachitis is so largely dependent in its osseous changes on a profound disturbance of nutrition that it can fairly be said to result from any disease which from its debilitating nature may interfere with the nutrition of the bones. In this way individuals whose nutrition has been seriously affected by hereditary syphilitis may develop rhachitis.

In regard to the actual lesions of the bones present in syphilis and rhachitis, there seems to be a concurrence of opinion that the pathological conditions are quite different. Thus, syphilitic bones very rarely present the spongy tissue peculiar to rhachitis, and rhachitic bones never show the osteophytes of syphilis.

Prognosis.—The prognosis in any case of hereditary syphilis is a serious one. In addition to the results which we are likely to have from the syphilis of the parents being early or late in regard to the impregnation, and from their having been thoroughly treated or not, there are certain facts to be remembered concerning the infant itself.

The prognosis is grave inversely to the number of weeks after birth when the disease first shows itself. The milder forms of the efflorescence justify us in giving a better prognosis than the more severe ones. In addition to these conditions which render the prognosis more favorable are the possibility of the infant being fed with good breast-milk or with a carefully prepared substitute food, and good hygienic surroundings.

The cases in which the spleen is much enlarged are evidently so profoundly affected by the secondary anæmia by which the enlargement is caused that the prognosis is almost invariably bad, and the degree of splenic enlargement may almost be taken as an index of the severity of the disease.

The opinion which we give to the parents should, however, always be very guarded, as, even though the disease may for the time apparently be entirely cured, it is always liable to appear again in later childhood and at puberty. When the disease is amenable to treatment these secondary symptoms almost always disappear by the second year, and in quite a large number of cases, where proper treatment has been thoroughly carried out, the infant recovers entirely and is as well and strong as though it had never had syphilis. In another set of cases, however, although the disease is apparently eradicated, in later years it is found to have left its marks in disturbances of the different functions and in the general lack of vigor of the various tissues.

Treatment.—The treatment of hereditary syphilis is first to adapt at once as nourishing a food as is possible to the infant's digestion. A healthy mother with plenty of good breast-milk will, as a rule, provide the best food for her infant.

If the mother's nutrition is reduced by syphilis or by any other chronic disease, the infant should be fed on a properly adjusted substitute food, while the general hygiene, such as fresh air, sunlight, and warmth, should be carefully regulated. A wet-nurse should not be employed unless she has herself had syphilis, in which case the same rules will apply to her nursing as to that of the syphilitic mother.

It should be remembered that the secretions from a syphilitic infant's mouth are very infectious, whether the disease is of the hereditary or of the acquired form. If, therefore, the mother is not syphilitic and the infant has acquired in any way a syphilitic lesion, the nursing must be discontinued and the infant fed on a substitute food.

The only drug which can be depended upon in the treatment of the early lesions of hereditary syphilis is mercury. This drug naturally would be employed from our experience with it in acquired syphilis, where it is more valuable in the early stage of the disease than at any other period. In like manner iodide of potash is of little use in the early stages of hereditary syphilis, while it becomes useful in the retarded form, which corresponds to the later stage of acquired syphilis.

It is important carefully to adapt the form of mercury which is given to the syphilitic infant according to its special idiosyncrasy for the drug, and also to regulate the means of its administration according to the necessity of having it act quickly, as is indicated in the more severe forms of the disease, and according to the sensitiveness of the individual's stomach or skin. Thus, mercury may be administered either through the mouth or through the skin. In the latter case it may be applied directly in the form of liquid or ointment or by means of subcutaneous injections. The last method should be used in very urgent cases only, for the tissues and skin of the syphilitic infant are especially liable to be irritated to such an extent that sloughing may take place, and the tissues under these circumstances are readily destroyed. When used, it should be in the form of corrosive sublimate.

The corrosive sublimate should never be given subcutaneously in larger doses than 0.0006 gramme ($\frac{1}{100}$ grain). Where the mercury is to be applied directly to the skin it may be in the form of corrosive sublimate baths, 0.3 to 0.6 gramme (5 to 10 grains) to each bath once daily, but practically it is found better to introduce it into the system by means of an ointment. This ointment may be the official mercurial ointment, either in full strength or diluted with some simple ointment, and this may be applied by means of inunction, as is the custom in the acquired syphilis of adults. After the infant's skin has been thoroughly washed, a small portion of the ointment should be applied to its back and rubbed carefully and gently into the skin for ten minutes. On the next day the same procedure can be carried out on the front of the chest; on the third day in the axillary regions; and on the following days respectively on the outer surfaces of the arms and thighs. I have found that the most practical way of applying inunctions to these infants is, after having thoroughly washed the abdomen, to spread the ointment thickly on a piece of thin soft flannel cut so as to reach from the ensiform cartilage to the pubes and to extend around the entire abdomen. This ointment is made in the following way:

PRESCRIPTION 78.

Metric.	Gramma.	Apothecary.	
℞ Unguenti oleati hydrargyri,		℞ Unguenti oleati hydrargyri,	
Unguenti lanolini............	āā 60|00	Unguenti lanolini............	āā ℥ii.
M.		M.	

The band should be allowed to remain in place for forty-eight hours. It should then be removed, and, after the skin has been thoroughly washed with warm water and soap and dried with a soft towel, the flannel should again be spread with the ointment and reapplied.

In giving mercury by the mouth I am in the habit of using the official hydrargyrum cum creta. I usually begin with 0.06 gramme (1 grain) of the drug, administered three times in the twenty-four hours. Within a few days the dose is increased to four times in the twenty-four hours, and if no unfavorable symptoms appear it is again raised to 0.12 gramme (2 grains) three or four times in the twenty-four hours.

The unfavorable symptoms which have just been referred to as possibly being caused by the drug are represented by diarrhœa. We must remember that the infants whom we are treating for hereditary syphilis are so young that the salivary secretion has been very slightly developed, and that therefore we naturally do not salivate an infant of this age so readily as we would a child or an adult. We must not, however, think that we can be guided as to the amount of mercury we are introducing into the infant's stomach by salivation, which is usually relied upon to indicate the physiological action of mercury. I have found it a safe rule to continue with the mercury until diarrhœa is caused, when the drug can be reduced in quantity, or even be omitted for a few days. When the intestine has become less sensitive we can again begin with a smaller dose, and one which by experiment has been shown not to cause diarrhœa in the especial infant.

Other forms of mercury, such as calomel in doses of 0.006 gramme ($\frac{1}{10}$ grain) three or four times daily, may be given by the mouth in these cases.

These various forms of mercury should be tried when for any reason one of them is found not to suit the case.

For the treatment of the fissures which occur around the lips and the lesions of the mouth, as well as those which occur at the anal orifice, I am in the habit of using a simple powder of calomel, which is dusted on the part affected. The mouth should be carefully cleansed several times during the day and a wash of chlorate of potash used at least twice a day. In some cases, although rarely, nitrate of silver is needed as an application to the ulcers when they are intractable. When there are crusts around the lips and in the neighborhood of the fissures, or where anal condylomata are present, the mercurial ointment just spoken of is of much benefit. The crusts should be carefully removed from the nose and this same ointment gently applied to the lesions. The application of this ointment to the abdomen is at times followed by an eczematous irritation of the skin of the abdomen. Under these circumstances any simple emollient should be applied in place of the mercurial for a few days until the skin has recovered, and the mercury can then be further diluted with lanoline or some simple ointment and reapplied, thus finally adjusting the strength of the mercurial to the vulnerability of the infant's skin.

In addition to the mercurial treatment, tonics in some form, especially iron, are at times required. It is usually in the later stages of the disease that they are indicated, and in cases in which the persistence of the splenic enlargement shows the presence of profound secondary anæmia.

After all the symptoms of syphilis have disappeared and the infant is entirely well, the mercurial treatment should be continued for some months, and also later during the first three or four years of its life, at intervals of three or four months, even when there is no return of the syphilitic symptoms. It should likewise be given at intervals during the period of the second dentition, and again at puberty. This treatment is especially important whether the infant appears to be in good health or not, as it tends to prevent a recurrence of the disease, and we should remember that a recurrence often proves very intractable to treatment.

The following cases illustrate the different phases of hereditary syphilis and the different conditions which are liable to be met with in this disease.

The first infant was three weeks old. Its mother looked well and strong, denied having had any miscarriages or disease of any kind, and asserted that the father was also healthy. Both of these statements were probably untrue, but an excellent opportunity for making a diagnosis simply by inspection and by a physical examination was given.

At birth the infant was puny and atrophic. It soon began to have occlusion of the nares. When one week old, an efflorescence of papules appeared on its arms, legs, and feet, with pustules on the palms of the hands and the soles of the feet. It did not vomit. The fæcal movements were of a good color and fairly well digested. The heart and lungs were normal. The splenic area of dulness was slightly increased, but the spleen could not be felt. There were marked fissures at the angles of the mouth, a muco-purulent discharge from the nose, and crusts forming on the eyebrows. The mouth and throat showed nothing beyond a pronounced erythema. There were papules and pustules on the body, and a squamous as well as a pustular efflorescence on the palms of the hands and the soles of the feet. There were maculæ on the buttocks. The anus showed nothing abnormal. The temperature was normal. The infant looked fairly well nourished.

There could be no question about the diagnosis in a case like this, and the statements of the mother regarding herself and her husband were entirely ignored, for by simple inspection it was clear that it was a case of hereditary syphilis.

The next infant was six months old. The mother, a healthy-looking woman with plenty of breast-milk, nursed the infant. She had had one miscarriage, in the third month, and this was her first child. The father denied having had any venereal disease.

At birth the infant was rather atrophied and had a general papular efflorescence all over it, and later a squamous efflorescence on the palms of the hands and the soles of the feet. It always had marked occlusion of the nares (snuffles). The infant was immediately placed under treatment, and at six months looked well nourished. It was a case of hereditary syphilis, and showed the beneficial results of good breast-milk and mercury, for it was very large for its age and was fat and strong-looking. It had, however, certain lesions of the bones which were the result of the syphilitic manifestations which it presented at birth. One of these lesions was represented in the marked prominences on either side of the frontal bone, with a somewhat depressed sulcus between them.

The first phalanx of the left little finger and that of the left third finger were swollen and somewhat reddened, and the tissues had a tendency to break down. This is the condition which has already been described as *syphilitic dactylitis*. Cases of tuberculosis of the bone often simulate this condition, and, in fact, so nearly approach it in appearance that the two diseases cannot be distinguished by simple inspection. The diagnosis must be made by considering the other symptoms.

The syphilitic infant is described essentially as atrophic; this is, as a rule, the case only when it is deprived of good breast-milk or of a properly proportioned substitute food, the atrophy being usually a fault in diet, provided that the intra-uterine nutrition has been good.

Syphilis is so prolific a source of miscarriage that a history of miscarriage in the mother justifies us in looking with suspicion on a doubtful lesion of the skin in her infant. A woman may have a number of miscarriages caused by syphilis, and may then, if she has been treated with mercury, give birth to a living syphilitic infant, or to one that is healthy. These facts are important for us to remember when we are considering the prognosis in a case of hereditary syphilis.

The next infant illustrates one of the many unusual forms of syphilis which may manifest itself in infancy.

A male, four months old, was brought to my clinic with syphilis of a rather aggravated type, and among other lesions with condylomata at the anal orifice.

It had a general papular efflorescence on the face, body, and limbs, including the palms of the hands and the soles of the feet. The left arm hung helpless by its side. The left leg was also somewhat affected. On examining the arm there was found a small, hard, painful, circumscribed swelling at the lower end of the humerus. No crepitation was detected.

The condition was one of the osseous lesions of syphilis, an *osteochondritis* accompanied by periostitis, which caused so much pain on movement as to disable the limbs and simulate both paralysis and fracture. Mercury was given and the infant recovered.

The next case is of remarkable interest, owing to the form and appearance of the efflorescence, which, although unusual, is so characteristic that it could represent no other disease than syphilis.

The infant was six weeks old. The mother stated that she had been married about three years, had had two children, and had had no miscarriages. She said that the father was well and strong, and that neither of them had had any efflorescence on their skin. The older infant was fourteen months old, and was healthy. The younger infant was being nursed by its mother. At birth it was apparently healthy and well nourished. Its skin was clear, its body fat, and there was no occlusion of the nares. This condition continued until it was eight days old. It then began to have occlusion of the nares (snuffles), a slightly hoarse voice, and an efflorescence on various parts of the body and limbs. This efflorescence consisted mostly of maculæ, many of which were circumscribed by healthy skin. They varied in size from 0.6 to 1.25 cm. (¼ to ½ inch).

Plate X., facing page 610, shows a number of lesions represented in this case.

In addition to the maculæ, which varied from a delicate pink to a yellowish-white color, there was a pustule on the outer side of the leg just below the knee. On the inner edge and almost on the back of the foot were the remains of a bleb which had broken down and had been emptied of its contents. There was also on the inner side of the foot, nearer to the heel, a small ulcer. The entire skin of the heel was reddened and had a shining appearance. The erythematous lesions in places on the leg were surrounded by normal skin, presenting a mottled appearance, and there were white spots on the skin. These latter, however, were caused merely by the peculiar distribution of the syphilitic maculæ.

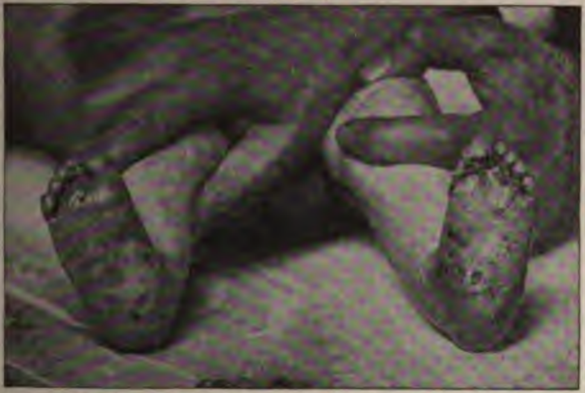
In addition to these lesions on the leg there were a few ulcers on the buttocks, and in addition to the maculæ on the soles of the feet there were some on the palms of the hands. A few scales showing a squamous condition could be seen on the left leg, but this lesion was not a prominent one.

The eyes were not affected. There were a few fissures about the mouth, but no lesions of the buccal mucous membrane, and there were no gummata around the anus.

The treatment of this case was by inunction with the oleate of mercury ointment and by the administration of hydrargyrum cum creta.

Fig. 120 represents the lesions of syphilis on the soles of the feet in a male. The lesions consisted of a number of bullæ, some of which had burst, and the tissue beneath having broken down, ulcerations were formed. There were also a few papules, some smaller bullæ, and some pigmented areas.

FIG. 120.



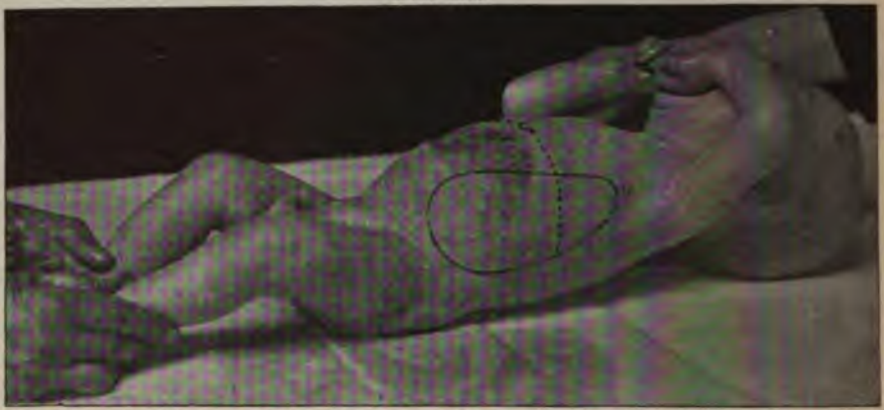
Syphilitic maculæ, ulcers, and bullæ on the soles of the feet. Male, 2⅓ months old.

This same case had the "waxen" pallor of the skin, so characteristic of the higher grades of grave anæmias. There was moderate enlargement of the liver, which on palpation was found to be hard and somewhat tender. The inguinal glands were slightly enlarged. The post-aural glands were enlarged. The spleen was much enlarged and extended, as indicated in Fig 121 by the black line, from the fifth rib to the left inguinal region. It was hard, but was not tender. There were no other glandular enlargements. The examination of the blood was as follows:

	Nov. 17.	Nov. 20.
Erythrocytes	3,387,000	3,300,000
Hæmoglobin	47 per cent.	45 per cent.
Leucocytes	20,000	20,000

There was a considerable variation in the size of the erythrocytes, which were pale in color. There was poikilocytosis in a moderate degree; there were also some microcytes and megalocytes. The mononuclear elements predominated (about three-quarters). The eosinophiles were not numerous.

FIG. 121.



Male, 2½ months old. Congenital syphilis. Grave secondary anæmia. Lower border of ribs, fifth rib, and enlarged spleen marked in black.

Late Manifestations of Hereditary Syphilis.—SYMPTOMS.—The manifestations of hereditary syphilis which appear at birth usually develop in the first three or four months of the infant's life. In certain cases of syphilis which are without doubt of the hereditary form, either no symptoms whatever are noticed in the early years of life, or they are so slight, or so lacking in the characteristics of syphilis, that it is sometimes impossible to recognize them as syphilitic lesions. The lesions of this late hereditary form correspond to the tertiary lesions of the acquired form. They appear in different periods of childhood or at puberty. These periods correspond to the time when a fresh outbreak of an attack of syphilis which has occurred in the early months of life is apt to take place. This is significant as leading us to suspect that the early symptoms of the disease have been overlooked rather than to believe that they did not occur.

Bones.—The lesions of the bones hold a prominent place in these later manifestations of hereditary syphilis. These lesions may be in the form of a periostitis, or an actual necrosis of the bone may take place either in connection with a dactylitis or with a simple lesion of the osseous tissue in any of the bones.

As these later forms of hereditary syphilis merely represent the same conditions which are met with in tertiary acquired syphilis, we should expect the most varied lesions. In this late form of hereditary syphilis the bones of the nose are frequently involved, and a flattening of the bridge of the nose is not uncommon. The cranial bones show certain alterations which at times are quite characteristic. The frontal bone may present a prominence on either side, which, with a depression more or less deep between the prominences, causes such a peculiar conformation of the head as to be almost characteristic of syphilis. This is well represented in Fig. 124, on page 541. In addition to these frontal prominences, at times there is a prominence of the centre of the frontal bone, which, with the apparent flattening on either side, causes a peculiar shape simulating the keel of a ship. Sometimes protuberances similar to those which have been described of the frontal bone may appear on the parietal bones. When they are bilateral the sagittal suture appears as a depressed sulcus between them, and this deformity of the skull, from its resemblance to the shape of the nates, has been designated by Parrot as the *natiform* skull.

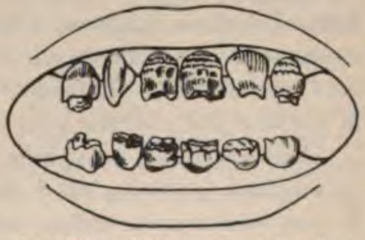
These tuberosities on the skull may also appear upon the long bones, either in the diaphysis or in the epiphysis. When the tibia is affected there is often so marked an increase in parts of the shaft of the bone, especially its middle third, that, as the enlargement is chiefly in the anterior portion, the swelling when prominent gives an appearance of curvature to the bone. This is, however, only a seeming curvature, as the posterior portion of the bone is not affected.

Mental Development.—An interference with the growth of children who are affected by these various osseous lesions of syphilis is not uncommon. There is frequently a lack of development, which shows itself usually in a failure of the individual to attain the ordinary height. The mental development is retarded, the children often appearing to be a number of years younger than they really are. This condition Fournier has designated as *infantilism.*

Teeth.—The first set of teeth in infants with hereditary syphilis have nothing characteristic about them; they show a lack of nutrition, a condition which may arise from many other morbid processes.

The second set of teeth, however, present certain characteristics. These characteristics are shown especially in the two middle upper incisors, in which the cutting edge of the tooth is worn away, leaving a convex surface with the convexity upward. The teeth are also apt to be somewhat far apart, and, as the child grows older, to assume a peg shape. The especial characteristics of syphilitic teeth were first described by Hutchinson. This peculiar shape of the teeth is not always present in syphilis, but when it appears it is certainly very suggestive of the disease. As was pointed out by Coleman, the dentist who examined Hutchinson's cases, in nearly every one of them there was a deficiency in the superior alveolar arch at the anterior portion, so great in some cases that when the jaws were closed the upper and the lower incisiors did not come together.

FIG. 122.



Syphilitic teeth of the second dentition.

Fig. 122 represents twelve syphilitic teeth of the second dentition. They are all more or less disorganized in a way which might occur from any cause which would interfere with the normal development of the teeth and cause their early decay. The middle two and the left lateral upper incisors show the notched and somewhat peg-shaped condition which is supposed to be characteristic of syphilis.

Nails.—The *onychia* which occurs as one of the earlier manifestations of hereditary syphilis has already been described. In the late form of syphilis another form of onychia is met with, characterized, according to Post, by a swelling at the base or the side of the nail, which becomes thickened, fissured, and brittle, with more or less deformity of the phalanx.

Eye.—In the late form of syphilis a peculiar inflammation of the cornea at times appears. It usually begins with a cloudiness of the substance of the cornea, with ciliary congestion. The entire cornea in this way becomes clouded. The affection is not accompanied usually by pain, and does not show any special congestion of the conjunctivæ. Hutchinson says that it is always symmetrical, although at first it is apt to begin with one eye and later to attack the other. The interval between the two attacks may extend over several years. This disease is called *interstitial keratitis*, and may for a few weeks seriously interfere with the sight. It usually disappears under treatment without leaving any trace of the disease behind it. On the other hand, opacities are sometimes left which interfere with vision. The total duration of the disease varies from six to eighteen months. Interstitial keratitis, according to Post, occurs most frequently in female subjects, and is most common between the ages of ten and fifteen, although it may occur much earlier, and, according to Fournier, may even be met with at birth. Complications may arise in the shape of iritis, choroiditis, and retinitis.

Ear.—Disturbances of hearing may occur from a number of causes, especially from those secondary to diseases of the pharynx. An especial form of *deafness*, however, without any special lesions to explain it, occurs in the syphilis of childhood, is usually intractable to treatment, and persists into later life. Extensive ulcerations produced by syphilis may occur in the *nose* and *pharynx* at any time during childhood.

Nervous System.—Syphilis of the nervous system may be congenital or acquired, involving either the brain or cord. It is very rare in children. It may occur as a diffuse inflammation of the meninges, as localized gummata, or as an endarteritis. Syphilitic meningitis and endarteritis present essentially the same symptoms as in adults. Gummata of the brain or cord present no symptoms in themselves to distinguish them from other cerebral tumors (see page 978). The lesions in the cord are, however, apt to be widely distributed, involving the cervical, dorsal, and lumbar regions, with preservation of some of the functions and complete loss of others. The rapid diminution of some symptoms and the persistence of others are characteristic. The presence of syphilis elsewhere and the marked improvement under antisyphilitic treatment are important points in the diagnosis. Syphilitic endarteritis and multiple gummata of the base have been observed in infants as early as fifteen months.

TREATMENT.—The treatment of the lesions which usually occur in the retarded form of syphilis is essentially with iodide of potash, either alone or in combination with some mercurial. The iodide of potash should be given at first in doses of 0.12 or 0.18 gramme (2 or 3 grains), and this dose should be gradually increased to 0.36 or 0.6 gramme (6 or 10 grains), or even more, as children often tolerate this drug remarkably well, and large doses are usually indicated.

When iodide of potash is given in combination with mercury, it is well to begin with corrosive sublimate in doses of 0.0006 gramme ($\frac{1}{100}$ grain) and gradually to increase the dose. Corrosive sublimate is, however, so apt to cause disturbance of digestion that I prefer to treat these cases by giving the iodide of potash uncombined with any other drug, by the mouth, and by applying the mercurial ointment to the skin.

The treatment of these later manifestations of syphilis must often be continued for long periods.

The following case illustrates the retarded form of syphilis, and shows the importance of carefully reviewing the previous history not only of the child, but also of its parents.

The child was a girl, thirteen years of age. The mother had had no other children nor any miscarriages. She had always been well, and had never shown any manifestations of syphilis.

The father, so far as could be ascertained, until recently had always been well and strong, and had shown no signs of syphilis. About one year previous he began to have cerebral symptoms, which rapidly increased, were accompanied by paralysis, and were undoubtedly of syphilitic origin.

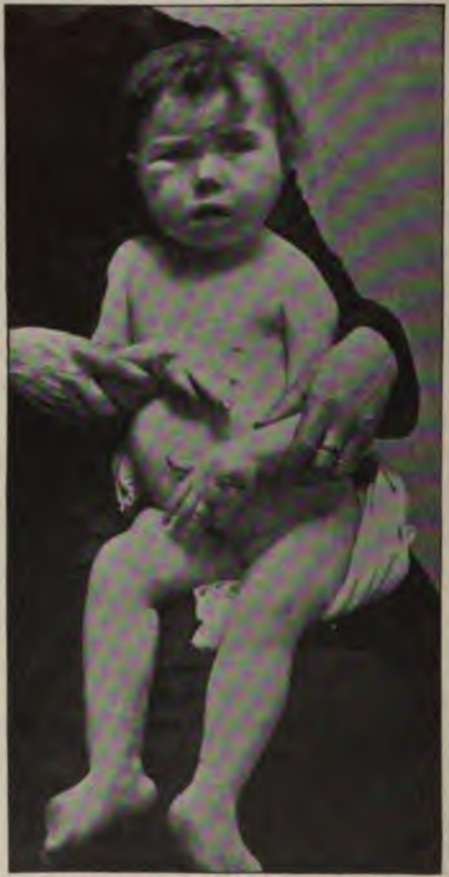
I was first called to see this child when she was suffering from a mild attack of appendicitis, which did not come to operation. At that time I noticed a peculiar conformation of the upper incisors, which made me at once suspect a case of hereditary syphilis. On further inquiry I learned that she had been treated some years earlier by an oculist for keratitis. The upper incisors were abnormally far apart and stunted in their growth. They were notched, as was also the left lateral incisor, which was peg-shaped and by its clearly cut notch represented more nearly than the others the characteristic syphilitic teeth. The right upper lateral incisor had a peculiar shape, the crown of the tooth coming down almost to a point. The other teeth were in many places deprived of their dentine, and were in various stages of disorganization.

On recovering from the appendicitis the child remained in a weak condition during the following year, looked sallow, and had continual headaches, which did not improve under the usual remedies. Treatment with iodide of potash was not only followed by the disappearance of the headaches, but also resulted in a healthy appearance of the child, who became perfectly well.

The following case is an illustration of the various tertiary lesions of syphilis.

A girl, three and one-half years old, had certain lesions on the face, arms, hands, and feet, which were the result of congenital syphilis. When this child was born she was apparently healthy. When she was three months old she was noticed to have occlusion of the nares, and at that time she had an attack of bronchitis lasting for three weeks. It was said that no efflorescence was ever noticed on her skin. When she was seven months old her hands began to swell, and at fourteen months the tissues around the metacarpal bones of the little fingers of both hands became reddened and ulcerated and the fingers assumed the pyriform shape characteristic of syphilitic dactylitis.

FIG. 123.



Late manifestations of syphilis. Female, 3½ years old.

When the child was about sixteen months old the feet began to swell, and in certain parts, especially the metatarsal bones of the right foot, the skin became reddened. When the child was three years old pieces of dead bone began to come away from the hands, and this continued for some time. At the same age, swellings began to appear over the upper maxillary bones, and an extensive reddened and swollen condition of the tissues existed under the right eye. The fontanelles were closed. There were evidently a periostitis and an osteochondritis of the right arm, and there was also an enlargement of the left ankle, accompanied by ulceration on the outer side of the malleolus.

The child was treated with the combination of mercury and iodide of potash shown in the following prescription :

PRESCRIPTION 79.

Metric.	Gramma.	*Apothecary.*	
℞ Hydrarg. chloridi corrosivi	0|03	℞ Hydrarg. chloridi corrosivi	gr. ss;
Potassii iodidi	3|75	Potassii iodidi	ʒi;
Aq. destil.	60|00	Aq. destil.	℥ii.
M.		M.	

S.—2 c.c. (½ drachm) 3 or 4 times in 24 hours.

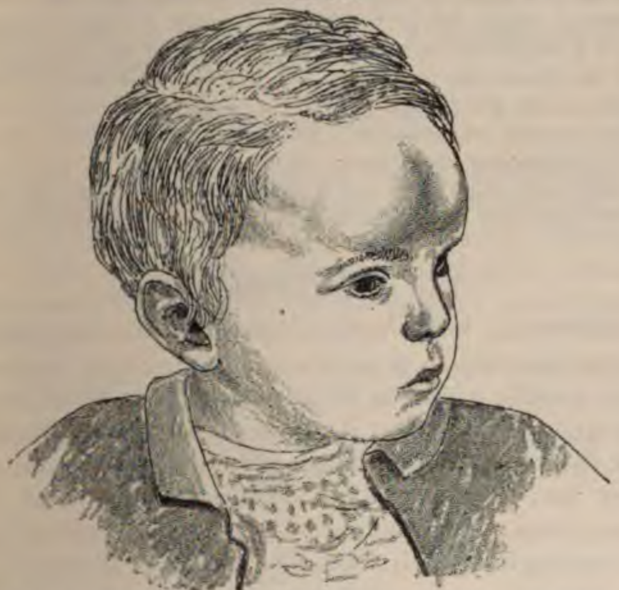
While it was taking this medicine all its symptoms abated, it seemed better and brighter, and the lesions showed a tendency to heal. Whenever the medicine was omitted all the previous symptoms returned.

The following case illustrates, among other interesting points, this same lesion of the bones.

The boy was six years old. He was rather pale, and had a somewhat peculiar frontal development, which well illustrates one form of syphilitic head.

There was a slight depression of the bridge of the nose and the bulging of the forehead on either side just above the orbital ridges. These prominences were accentuated by the deep sulcus between them, extending from the depressed nasal bones upward almost to the margin of the hair. This condition represents the typical syphilitic head.

FIG. 124.



Hereditary syphilis. Male, 6 years old. Abnormal prominences of frontal bone.

The boy was in fair health, and had nothing abnormal about him on careful physical examination. His mother brought him to the clinic to receive a course of treatment for a few months in order to prevent a recurrence of his infantile syphilis.

The boy had been treated at the Children's Hospital when he was six weeks old. The mother had been well and strong, and had never had any other children nor any miscarriages. The father had had a primary syphilitic lesion one year previous to the birth of the child, which was followed by secondary manifestations. The mother had plenty of good breast-milk, and nursed her infant until he was nineteen months old. He was never atrophic, and although pale was apparently well nourished. At birth he showed a bullous efflorescence of medium grade. During the early weeks of his life he did not receive any medical treatment, although he had a general efflorescence of macules, pustules, and bullæ. At about the fifth week he lost the use of his left arm. When seen at the sixth week he showed a number of lesions besides those described, and it was doubtful if he would live. These lesions consisted of fissures at the corners of the mouth, mucous patches in the mouth, condylomata of the anus, and occluded nares. There was not at that time the peculiarly formed head which is now present. The left arm was helpless and was supposed to be broken; in fact, there was some crepitation, and probably there was a slight separation of the epiphysis of the distal end of the humerus. There seemed to be considerable pain in the arm, which made the infant restless and fretful. Insomnia was a prominent symptom. The arm was put in a light splint, and the oleate of mercury ointment (Prescription 78, page 532) was ordered.

The infant was not seen for a week. When he was brought back to the hospital the right arm was found to be helpless, and the mother stated that the ointment had been discontinued, as it caused excoriation of the skin. The ointment was then reduced one-half with lanoline, and hydrargyrum cum creta was given three times daily in doses of 0.06 gramme (1 grain).

In three days he was much better, the paralysis soon disappeared, and nothing abnormal was detected about the arms. The hydrargyrum cum creta was increased to 0.24 gramme (4 grains), but as this caused diarrhœa the dose in a few days had to be reduced to 0.18 gramme (3 grains). In the course of the next month the nasal symptoms and the efflorescence had disappeared, and the infant seemed perfectly well.

Six months later he was brought back to the hospital with a return of the condylomata and a slight papular efflorescence. The same treatment as before was carried out. The syphilitic manifestations disappeared, and did not return.

He was kept under observation and treated from time to time for three or four years. The first teeth were cut at nine months, and were in fair condition.

THE EXANTHEMATA.

In contradistinction to the various diseases of the skin which dermatologists are accustomed to designate as exanthems of local origin are certain acute, specific, infectious diseases which are called the exanthemata. This group of infectious diseases is of especial interest in connection with children, as it is among children that they most frequently occur. They can, however, attack individuals of any age. Although none of these diseases are entirely self-protective, yet the instances in which they develop in an individual more than once are rare.

The exanthemata comprise five diseases,—*scarlet fever*, *measles*, *rubella*, *varicella* (*chicken-pox*), and *variola* (*small-pox*). Each of these diseases is characterized by certain conditions common to all. Besides being infectious, each disease runs a definite course and is self-limited, facts which should be remembered when we are studying its diagnosis and treatment.

The course of these exanthemata from the time when the infection takes place up to the appearance of their later manifestations may be divided into distinct stages. In the first of these certain micro-organisms are supposed to enter the system, and, so far as external appearances and general symptoms are concerned, to remain dormant for a time, constituting what is called the *stage of incubation*. This stage of incubation is followed by certain general symptoms resulting from the supposed development of the special organisms and constituting the *prodromal stage*. These prodromal symptoms are, after intervals varying according to the special disease, followed by an efflorescence on the skin, which marks the third stage of the disease, called the *stage of efflorescence*. The efflorescence in its turn is followed by what is called the *stage of desquamation*, this desquamation being more or less pronounced in proportion to the intensity of the lesions of the skin which have occurred during the stage of efflorescence.

Although in a large number of cases the diagnosis of these diseases can be determined by the appearance of the efflorescence and its location, yet instances occur not infrequently in which the efflorescence is very misleading. We should, therefore, be familiar with the characteristics of the other stages, for it is by carefully considering the pictures which they present to us as a whole that we are enabled to make a correct differential diagnosis of the especial case. Thus, a papular efflorescence, although significant in most cases of measles, may also be present in other members of the group, while an erythema closely resembling scarlet fever may occur in variola, measles, or rubella.

SCARLET FEVER.

Scarlet fever is an acute infectious disease, characterized by a short incubation, short prodromal stage, erythematous efflorescence, pronounced desquamation, and long course. The micro-organism which produces it has not yet been determined. With the exception of variola, it is the most dangerous of the group, and is therefore the most important of all the exanthemata.

The complications of scarlet fever are so much more serious and its sequelæ so much more common and grave than those of varicella and measles, that its immediate diagnosis and prompt treatment are of vital necessity in every community where numbers of children are liable to be attacked by the disease.

Etiology.—Scarlet fever is the most irregular of all the exanthemata in its virulence and in the manifestations which it presents in different individuals. It is usually epidemic, returning to the same localities after a period of years. It is at times sporadic, and is commonly endemic in large cities. The epidemics of scarlet fever vary in severity, so that we cannot ascribe the virulence of the disease in certain years to individual susceptibility. The sporadic cases may be of the most malignant or of the mildest type. A mild case may give rise to a malignant case in another child, and a malignant case may give rise to a mild one. The epidemics of scarlet fever spread slowly, in contradistinction to those of measles, which spread rapidly. Scarlet fever may occur more than once in the same individual, but this is rare. Instances have occurred in which a child has had scarlet fever, and, on returning after several weeks to the same room, even after it had been disinfected, has again contracted the disease in its typical form. The source and identity of the contagium have not been definitely determined. The bacterial infection is secondary, and is mostly from the streptococcus pyogenes. The skin appears to be the chief vehicle of transmission. It has, however, been shown that the discharge from the nose and throat, both in the early stages of the disease and when they are prolonged even after the stage of desquamation, may be a source of infection. The contagium has a great tenacity for clothing and other articles, and may be capable for many months of reproducing the disease.

In reference to what has been said concerning the slow spread of scarlet fever during epidemics in comparison with the rapid spread of measles, certain clinical facts are significant. The disease does not seem to be very infectious in its early stages. We are thus led to believe that it is during the stage of desquamation that the contagium is most likely to be disseminated. Measles, on the other hand, is known to be highly infectious in its early stages, and for this reason to spread more quickly.

Although the contagium of both diseases may be active through their whole course, yet the general rule is early infection in measles, late in scarlet fever. The following cases illustrate this conclusion:

A boy six years old and a girl four years old slept in the same room, with their beds touching each other. The boy was taken sick May 1, but remained in the same room with his sister during that day and the following night. He was seen by me early on the morning of May 3, and was then found to have scarlet fever. His sister was taken to the country, and the boy was left in charge of a trained nurse. There was absolutely no communication between the town-house and the country-house, either by people, clothes, or letter. I myself did not again see the boy during his sickness, having placed him under the charge of another physician.

On June 1 I was called to see the girl, and found that she had scarlet fever. There were no other cases of scarlet fever in the vicinity of the country-house where she had remained since leaving the city.

The boy at this time was desquamating freely, and four days previous to the girl's being taken sick a letter written by him had been sent to her, and she, after having had it read to her, had been allowed to keep it under her pillow.

A careful study of this case led to but one conclusion,—that the boy during the period of his desquamation had infected his sister at a distance of twenty miles by enclosing the contagium of scarlet fever in an envelope. The girl, although she had been in the same room with the boy for thirty-six hours at the beginning of the disease, and although susceptible to the disease, had not contracted it at that time, owing to its very slightly infectious nature in its early stages. On the other hand, the stage of incubation of scarlet fever being only a few days, and many instances having proved that the disease is very infectious during its period of desquamation, it was evident that the girl had been infected by means of the letter.

In the following year, on May 20, I was again called to see the same boy. He had been well in the morning, but in the afternoon was found to have a high pulse and temperature, with coryza and lachrymation, so that it was deemed best to send the sister, who had been in the nursery only a few hours with her brother after he had been taken sick, to another house, while the boy was absolutely isolated. Three days later the boy was found to have measles. Ten days later the girl was attacked by measles. This case merely emphasizes the now commonly accepted belief that measles, in contradistinction to scarlet fever, is highly infectious in the early hours of the disease.

Whether the contagium of scarlet fever can be carried by the breath is somewhat doubtful, but it is probable that any of the excretions may contain it, and that it is especially liable to be transmitted by milk, clothing, toys, books, carpets, and other articles. Scarlet fever may occur in certain animals, and the contagium may be transmitted by others, such as dogs and cats.

An instance which leads me to believe that scarlet fever may be transmitted at a very early stage of the disease is the following:

A child who had contracted scarlet fever a few days previously came to a party given in a small and practically isolated community. At this time the child was beginning to feel sick and to complain of a sore throat. A spoon which had been used by her was also used, before it was washed, by one of the other children. Six or seven days later this second child was attacked by scarlet fever.

A careful and critical investigation of the possible origin of the second case resulted in the evidence strongly pointing towards a direct transmission of the contagium from the mouth of one child to that of the other by means of the spoon.

Scarlet fever may occur at all ages, but is rare during the first year of life. It has been met with in young infants who were nursing, and who have proved to be the focus of infection for a whole household.

The following table was compiled by McCollom. It represents the age and the number of deaths in one thousand cases of scarlet fever treated in the contagious wards of the Boston City Hospital:

TABLE 65.

One Thousand Cases of Scarlet Fever, by Ages, with the Deaths.

Years.	Cases.	Deaths.	Years.	Cases.	Deaths.
Under 1 year	9	2	19 years	10	1
1 year	24	8	20 years	7	0
2 years	66	20	21 years	8	3
3 years	115	21	22 years	8	0
4 years	99	7	23 years	15	0
5 years	124	9	24 years	9	0
6 years	106	7	25 years	7	0
7 years	64	6	26 years	5	0
8 years	62	5	27 years	7	1
9 years	58	1	28 years	6	1
10 years	53	3	29 years	3	0
11 years	21	1	30 years	9	0
12 years	20	0	31 years	3	0
13 years	23	1	32 years	3	0
14 years	10	0	33 years	2	0
15 years	12	0	34 years	3	0
16 years	10	0	35 years	1	0
17 years	7	0	41 years	1	0
18 years	9	1	50 years	1	0
				1000	98

PATHOLOGY.—The organs primarily affected in scarlet fever are the *skin* and the *throat*. The principal complications which arise in the course of the disease are connected with the *ear* and the *cervical glands*. The chief sequela, and the only one which is at all common, is *nephritis*. *Cardiac disease* may occur, but is commonly secondary to the nephritis.

Lesions of the other organs are somewhat unusual and have no definite connection with the scarlet fever. They are generally due partly to the fever, partly to the septic processes which have arisen in the course of the disease, and are essentially those of acute inflammation with cellular exudation, and with focal necrosis of the liver and kidneys. The changes are a marked infiltration of leucocytes in the tongue and in the desquamating skin. The internal organs show an infiltration with plasma-cells, and this is especially noticeable in the kidney in the acute interstitial form of nephritis. A proliferation of cells is found in the follicles of the lymph-nodes, and these cells are also often found infiltrating the coats of the veins, seeming in many places only to penetrate the endothelium and forming plugs in some of the smaller vessels. Hyperplasia of the lymph-nodes is a constant and marked characteristic. As a rule, the spleen is enlarged and shows marked follicular hyperplasia. (Pearce.)

Skin.—Macroscopically the morbid conditions of the skin in scarlet fever, although varying in their manifestations, are usually represented by an intense general erythema covered thickly with minute macules, which are of a darker red than the accompanying hyperæmia. Minute white spots may also appear thickly scattered over the reddened surface, probably arising from areas of unaffected skin existing in the midst of the general hyperæmia. An appearance like that of milium is also at times noticed to be scattered on the areas of skin affected by the erythema. No evidence of this hyperæmic condition, which is so pronounced during life, is found after death.

According to Neumann, microscopic examinations of the skin by means of hardened sections of specimens from cases of scarlet fever and measles in the stage of desquamation explain in a measure why the former is so much more likely to be infectious during its stage of desquamation than is the latter. In contradistinction to the pathological processes which are found in the skin in measles, and which affect chiefly the blood-vessels and glands, a very different picture is presented on examination of sections of skin taken from cases of scarlet fever. In the latter we find the pathological process represented especially by exudative cells, which are very numerous and closely packed together, reaching even up to the horny layer of the epidermis. Occasionally these exudative cells may finally take the place of the epidermal cells, appearing on the free surface of the skin, and are gathered thickly among the excretory ducts of the cutaneous follicles. It is thus readily understood why the tissue proper of the skin and its epidermis present no marked changes in measles, and why the epidermal cells are far less likely to carry the contagium than in scarlet fever, in which the possibility of contagium exists until the desquamation has entirely ceased.

Throat.—The earliest lesions of scarlet fever appear on the mucous membrane of the hard and the soft palate. This appearance is very similar to the efflorescence which is seen on the skin, except that the minute white spots do not appear on the congested mucous membrane. Forchheimer states that the exanthem of scarlet fever appears from twelve to twenty-four hours before the efflorescence; it appears upon the pillars of the fauces in the form of the characteristic puncta, then rapidly spreads over the mouth in the form of a scarlet-red coalescing efflorescence, which finally ends in desquamation, producing the strawberry tongue, and lasting well into the second week of the disease. These pathological conditions which occur in the throat in scarlet fever may either be simply catarrhal, or result in one of the more severe inflammatory conditions affecting the tonsils, the pharynx, and the larynx.

The tonsils are uniformly and extremely bright red, and are thus to be differentiated from their dusky red color in cases of diphtheria before the membrane has appeared. On the hard and soft palate a punctate efflorescence is seen, and this appearance in from twenty-four to forty-eight hours may assume a yellowish color not ordinarily seen in catarrhal conditions of the throat or in diphtheria.

When a membrane is seen it is impossible without a culture to differentiate conclusively from diphtheria. In many cases, however, we can make a fair diagnosis by the color of the membrane in scarlet fever being whiter and the thickness less, as a rule, than in diphtheria.

As is stated by Delafield and Prudden, one of the most marked features of scarlet fever is the predisposition which it entails to the incursion of pathogenic germs other than those which we believe to cause this disease. Thus, in addition to the inflammatory lesions produced by the scarlet fever organism an acute exudative inflammation of the mucous membrane may occur, and may be associated with them. This is apparently caused by the growth of a streptococcus which, according to Welch, in morphological and biological character seems to be identical with the *streptococcus pyogenes*. In these cases there may be much or little fibrinous exudate, and there may be none at all in the early stages, or even through the whole course of the affection. The pellicle when formed may be more or less adherent, and sharply circumscribed, or it may tend to spread. The submucous tissue may show little change, or much congestion and œdema, or it may be the seat of suppurative inflammation. The entire process may be confined to the tonsils. While under these varying conditions the inflammatory process is usually a local one and runs its course, with or without the symptoms of septicæmia, occasionally the streptococcus finds access to the blood and may induce the lesions of pyæmia. On the other hand, it may by inhalation gain access to the lungs and induce varying phases of complicating broncho-pneumonia.

The staphylococcus pyogenes is not infrequently associated with the streptococcus in these lesions, but it is not apparently of great significance. Simulating very closely as it does in many cases both the local and the general phenomena of diphtheria, this pseudo-membranous condition was formerly confounded with it, but it is now recognized as a distinct disease.

There have been a number of extended investigations made on what are called the pseudo-membranous inflammations of the throat in scarlet fever. Booker has reported eleven cases of pseudo-membranous angina (two fatal) complicating scarlet fever, and one case of simple angina without exanthem in a family three members of which had scarlet fever. In all these cases, as well as in four scarlatinal anginas without pseudo-membranes, Booker found streptococci as the predominant organism, and in none was the Klebs-Loeffler bacillus present. The staphylococcus aureus was found in eleven cases without apparent influence on the severity of the disease. No difference was observed between the early and the late pseudo-membranous anginas in regard to the bacteria present. Booker describes with much detail the morphological and bacteriological characteristics of the streptococci found, and divides them into groups.

Park, in a series of one hundred and fifty-nine cases, reports nineteen cases of pseudo-membranous inflammation of the throat complicating scarlet fever. In seventeen of these cases streptococci predominated, and in only two was the Klebs-Loeffler bacillus present. Staphylococci were found in only a few cases. Williams has also reported cases of this kind, and Morse has reported ninety-nine cases of pseudo-membranous inflammation of the throat complicating scarlet fever. The Klebs-Loeffler bacillus was found in twenty-three, with a mortality of forty-three per cent., and was not found in seventy-six, with a mortality of twenty-one per cent.

Finally, we may conclude that in scarlet fever the mucous membrane of the throat is rendered peculiarly vulnerable to the invasion of pathogenic germs. When the morbid condition in the throat is represented by a pseudo-membrane it will be found that in the great majority of cases the process, as stated by Welch, is due to streptococci; but when diphtheria is prevalent and the opportunities are favorable for exposure, a large portion of the pseudo-membranous cases may be due to the Klebs-Loeffler bacillus.

In addition to the lesions of the throat just described, the micro-organism of scarlet fever may attack the nasopharynx. In this way, and by direct extension through the Eustachian tubes, secondary aural lesions may be produced. The morbid changes in the mucous membrane of the nasopharynx which thus take place may result in a thickening of the tissues, which in some cases lasts for many months after the scarlet fever has run its course.

Ear.—The pathological condition of the ear which is most commonly met with in scarlet fever is an acute inflammation of the middle ear. This inflammation is likely to result in destruction of tissue, the formation of adhesions, the establishment of a long-continued suppurative process, and an accompanying necrosis.

Cervical Lymph-Nodes.—There may be hyperplasia of the cervical lymph-nodes. This condition is sometimes accompanied by inflammatory œdema of the tissues of the neck, which may go on to suppuration and even to gangrene. In these cases streptococci are found in the glands and in the areas of suppuration. The infection is supposed to originate in the throat. The enlarged glands are, as a rule, indicative of secondary or mixed infection, although it is possible that the slighter forms of enlargement may be due to reflex irritation with resulting hyperplasia from the scarlet fever contagium. In the severe form the glands are at times very much enlarged, and when a gangrenous process results the blood-vessels may be affected to such an extent as to be ruptured.

Kidney.—In scarlet fever, as in a number of other infectious diseases, there are certain poisons produced in the course of the disease which are probably soluble in character. The results of bacteriological cultures in scarlet fever have shown that in a number of cases there is a general streptococcus infection, the infection probably coming from the lesions in the pharynx. In these cases of general infection streptococci may be cultivated from most of the organs of the body, there being a general septicæmia. In a number of these cases extensive lesions may be found in the kidneys, and yet these lesions may bear no relation whatever to the presence or absence of streptococci. In like manner, streptococci may be found in the kidney without any lesion of the kidney. These lesions are diffuse, and affect both kidneys and all parts of the kidney. From the best evidence which we have it would seem that the virus, or whatever it is which produces the lesions in the kidney, is not a living organism, but is a soluble chemical poison produced by the organisms of scarlet fever, or by other organisms located in some other part of the body. This soluble poison when produced elsewhere is taken locally into the blood and affects various parts of the economy. In post-mortem examinations of scarlet fever certain lesions will usually be found in the kidneys.

These lesions, according to Councilman, may be divided into two classes, (1) represented by simple degeneration of the epithelium, and (2) represented by marked changes in the tissues of the kidney.

In the ***first class*** of cases the soluble poison may only affect the integrity of the capsular epithelial cells of the glomeruli. The poison may produce certain degenerative changes in these, but need not be accompanied by any proliferation of cells, or by any condition which would be characterized as inflammatory. It is more than probable that these simple degenerative lesions are accompanied during life by evidence of albuminuria, and in case death takes place there may be no macroscopic evidence of any lesions in the kidneys. Careful microscopic examination, however, will show a condition of degeneration in the capsular epithelium of the glomeruli. Associated with this there will usually be found cloudy swelling of various degrees of intensity in the cells of the convoluted and the smaller collecting tubules. The degeneration here is rarely of a fatty character. Clinically, in the purely degenerative changes there may be only albuminuria with the presence of faint hyaline casts, and here and there a few leucocytes.

In the *second class*, owing to a greater intensity in the action of the poison, or to some possible difference in its character, more marked changes may take place in the kidney, and may be accompanied by the degenerative lesions which are distinctive of the first class. Different forms of lesions may occur in the second class, and, according to the predominance of one form over the other, may characterize a special form of renal disease. These lesions may be divided according to their anatomical distribution into *interstitial*, in which there is marked proliferation of the interstitial tissue of the kidney, and *glomerular*, in which the lesions are chiefly confined to the glomerulus and its capsule.

In the *interstitial* form there will be found in the interstitial tissue between the tubules accumulations of cells, the source of which is not clear, although they probably come from the blood. Most of these cells have the character of plasma-cells, but among them are a few lymphoid cells and polynuclear leucocytes. This form of nephritis should be considered as purely interstitial, since its lesions are in no way related to those of the epithelial tissue. There is both a general and a focal infiltration of cells in the interstitial tissue. The focal infiltration is found principally in the cortex of the kidney and about the glomeruli, the glomerulus frequently appearing as a centre from which the infiltration extends into the interstitial tissue between it and the surrounding tubules.

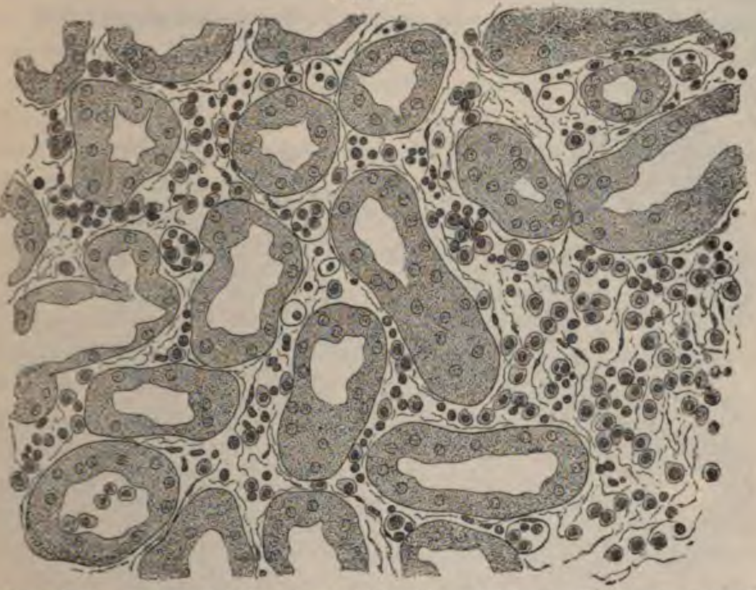
This form of nephritis was first described by Wagner as the lymphoid kidney. The kidney, macroscopically, is swollen; the capsule is easily stripped from the cortex, and is moist, whitish, and opaque. Usually there is no evidence of hemorrhage, although in some cases points of punctiform hemorrhage may be found in the cortex and in the intermediate zone.

Clinically, in this form there may be little evidence of the severity of the lesions. There may be, however, albuminuria corresponding to what is seen in the purely degenerative class. The quantity of the urine may be very little diminished, and casts may be present, as well as a certain number of desquamative epithelial cells and leucocytes.

These lesions are not confined to scarlet fever, but may be found in diphtheria, in measles, and in other infectious diseases of children, but they are not common in the infectious diseases of adults.

Fig. 125 represents a microscopic section, made by Councilman, and is a good example of these interstitial lesions in scarlatinal nephritis. The section was taken from a case of pure scarlet fever. There was no anuria and no dropsy. The kidneys were enlarged, whitish, and without hemorrhage. Cultures from this case gave a general infection with streptococci in all the organs except the kidney, and yet the kidneys, notwithstanding the extent of their lesions, were found to be free from streptococci. The epithelium of the tubules was somewhat swollen. The tubules

FIG. 125.



Interstitial nephritis. Section of kidney from child with scarlet fever. (Hartnack, ocular No. II., objective No. VIII. Tube closed.)

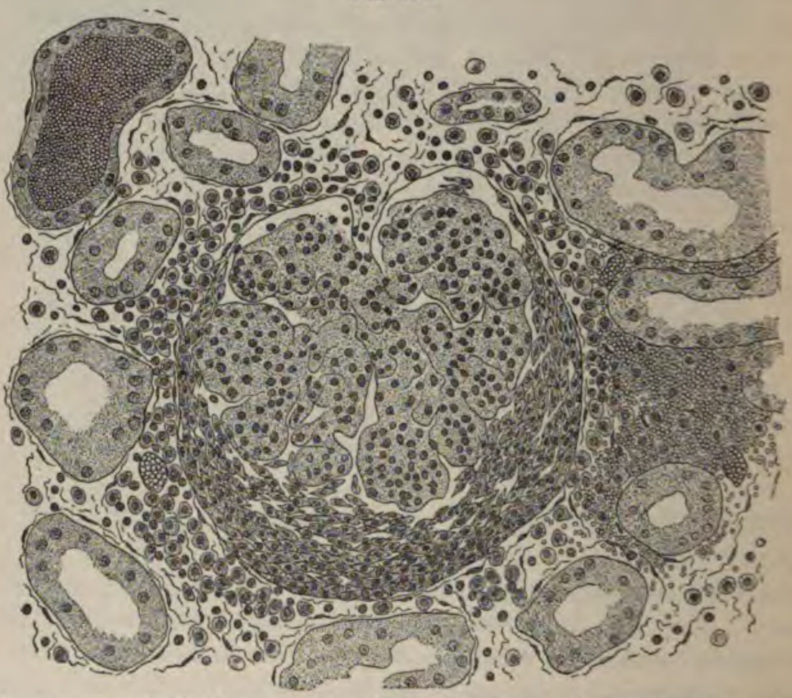
themselves were slightly dilated, and the epithelium was more granular than normal. The interstitial tissue was much more extensive than normal. The spaces between the tubules were increased both by œdema and by cellular infiltration. In the interstitial tissue blood-vessels were seen filled with cells of the same character as those outside. It is probable that most of the cells outside the vessels came from proliferation of the cells of the blood-vessels. The round spaces in the interstitial tissue represent blood-vessels.

Another form of nephritis, called the *glomerular*, is much more frequently found in scarlet fever than in any other of the acute infectious diseases of children, but is not so common as the acute interstitial form. In this glomerular form the chief lesion of the disease consists essentially in a proliferation of the capsular epithelium combined with hyperplasia of the connective tissue.

The proliferation of the capsular epithelium leads to the formation of masses of cells within the capsule between the glomerular capillaries and the capsule. These cells evidently result from the proliferation of the capsular epithelium. As a result of this there may be greatly increased pressure on the vessels of the glomerulus, possibly with an obliteration of these vessels. The cellular infiltration in the interstitial tissue is not so extensive as in the other form. Accompanying these changes in the glomerulus there is almost always more or less hemorrhage both in the tubules and in the interstitial tissue.

Fig. 126 represents a section made by Councilman of glomerular nephritis. This section was taken from a case of scarlet fever complicated by glomerulo-nephritis. In the centre of the field a glomerulus is seen, with an infiltration of cells in the capsular space. The capsular cells are oval and distinctly epithelial in character. Cellular proliferation of the cells having generally the character of those in the section of interstitial nephritis (Fig. 125) is to be seen in the interstitial tissue. In the tubule at the left upper corner there is evidence of hemorrhage, and some hemorrhage is also to be noticed in the interstitial tissue on the right of the specimen. In this case the anuria and dropsy were extreme.

FIG. 126.



Capsular glomerulo-nephritis. Section of kidney from child with scarlet fever. (Hartnack, ocular No. II., objective No. VIII. Tube closed.)

This form of nephritis may be best designated as *capsular glomerulo-nephritis.* The kidney is swollen and much more hyperæmic than in the interstitial form. The markings of the cortex either are obscured or cannot be made out at all, and there are numerous areas of hemorrhage and hyperæmia, giving the kidney a mottled appearance.

It is this capsular glomerulo-nephritis which gives the most marked clinical evidence of the extent of the lesions in the kidney. In this form dropsy is almost always present, the amount of urine is greatly diminished, and in the more severe cases there may be complete anuria. Blood-casts are found more frequently in the urine than in the interstitial form. The diminution in the amount of the urine points to involvement of the glomerulus. Even severe cases of this form may be recovered from. The process of cell-proliferation may cease, the cells formed in the capsular space may disappear and pass out, and the kidney in after-years may show few or no evidences of the process through which it has passed. In a certain number of cases, however, from this form of nephritis a chronic nephritis is developed. Cases of this kind have been reported, notably one by Aufrecht. .

In both the interstitial and the capsular glomerulo-nephritis fatty degeneration of the epithelium is not found to any degree. The epithelium is frequently swollen and granular, and may be hyaline.

These two forms of nephritis should be separated from each other, although transitions between their lesions are found. Usually they can be distinguished microscopically.

We can, therefore, recognize three pathological conditions of the kidney in scarlet fever: first, *the purely degenerative;* second, *the acute interstitial;* and third, *the capsular glomerular.*

Heart.—The pathological conditions of the heart which are at times found in scarlet fever do not differ in their macroscopic appearances from those met with in other diseases. Cardiac disease occurring in the course of scarlet fever may arise in two ways: (1) from the general septic condition existing during the period of the height of the temperature and general efflorescence, and represented usually by an endocarditis; (2) at a much later period from a nephritis which has arisen as a complication, and following which, from the resulting increased blood-pressure, enlargement of the heart has been produced, which may be represented by hypertrophy or by dilatation, or by both.

In connection with this subject, Silbermann has found on examining a large number of cases of nephritis during attacks of scarlet fever a decided hypertrophy of the heart combined with dilatation. In some cases both sides of the heart were equally affected, but usually only the left side was involved. In only a few cases was there found a partial fatty degeneration of the muscular fibres; the endocardium, pericardium, and blood-vessels were normal. According to Silbermann's observations, the cardiac affection was related to the post-scarlatinal nephritis, and not to the scarlet fever process itself, as the hypertrophy was never found when the child died in the early weeks of the scarlet fever. He calls attention to the short period which intervened between the first appearance of the nephritis and the consecutive heart hypertrophy, in many cases the time not being much longer than a week. He also noticed that in the cases in which hypertrophy and rapid dilatation followed the acute nephritis of scarlet fever the ages of the children were three and a half, four, five, and six years, and the post-scarlatinal cardiac enlargement corresponded to the physiological hypertrophy referred to on page 91.

Incubation.—The stage of incubation of scarlet fever is uncertain and irregular, but, as a rule, it is shorter than that of any of the other exanthemata. It is usually less than seven days, and quite frequently it is only from two to four days.

Variations in the Type of Disease.—Scarlet fever may be divided into two forms, the *benign form* and the *malignant form*. The difference in the symptoms of the common, or benign, form of the disease from those of the rare, or malignant, form is very striking. They could well be classified as entirely separate diseases, were it not that the contagium has been proved to be the same in each, by the fact that one form of the disease may give rise to the other in different individuals. It seems as though it were more the susceptibility of the individual to the scarlet fever contagium than the contagium itself which produces a greater or less severity of the symptoms.

BENIGN FORM OF SCARLET FEVER.

The benign form of scarlet fever either runs a simple typical course or is accompanied by variations and complications, which makes its course irregular.

Symptoms.—*Prodromata.*—The invasion of the disease is usually sudden and, as a rule, active. The child feels very sick, looks dull, complains of sore throat and nausea, and in a large number of cases vomits continuously. The vomiting usually ceases in the stage of efflorescence and often before the prodromal stage has ended. The pulse is rapid. The temperature is high,—39.4°, 40°, 40.5° C. (103°, 104°, 105° F.). In infants and very young children if the temperature rises to 40° or 41.1° C. (104° or 106° F.) convulsions are very likely to occur. The higher the temperature at the beginning of the disease the more active the symptoms, and the shorter the prodromal period the more severe will be the case. An initial temperature of 40° C. (104° F.) points towards a severe case.

Young children seem to show a less sensitive condition of the throat than is met with in older children and in adults. The appearance of the mucous membrane of the throat, although perhaps not characteristic, as at times a simple non-infectious pharyngitis may simulate it quite closely, is, in connection with the general symptoms, at least suggestive. The mucous membrane of the hard and the soft palate and of the pharynx is much congested. On the hard and the soft palate thickly scattered over the reddened surface are minute macules the color of which is a little darker red than that of the intervening mucous membrane. This condition represents the earliest stage of the efflorescence which later appears on the skin.

The length of the prodromal stage varies, as a rule, from twelve to thirty-six hours. During this stage the temperature continues to rise somewhat, and at its end the efflorescence appears on the skin.

Efflorescence.—The efflorescence of scarlet fever is of an erythematous and punctate character, sometimes looking as though minute macules had been sprinkled over the general redness of the skin. It starts on the front of the neck and the upper part of the chest, and rapidly extends all over the body and extremities, and upward to the face. This characteristic order of invasion of the skin aids us in distinguishing the efflorescence of scarlet fever from that of the common erythema which occurs in such diseases as pneumonia, and in cases in which certain drugs, such as belladonna, have affected the skin and the efflorescence comes out everywhere at once and has an irregular distribution. It also enables us to distinguish the disease from measles, in which the efflorescence begins on the sides of the neck and on the face and extends downward. On gently drawing the finger over the efflorescence of scarlet fever the resulting white mark remains longer than is the case with a common erythema. The efflorescence of scarlet fever continues to extend over the body for two or three days after its first appearance. During this period the tongue is much reddened and its papillæ appear very prominent, constituting what has been called the "strawberry tongue." This condition is to be distinguished from the enlarged papillæ seen at an earlier stage of the disease on the tip and sides of the tongue. McCollom, in a study of one thousand cases, describes this latter condition as diagnostic of scarlet fever. There is at times in this stage great irritation of the skin.

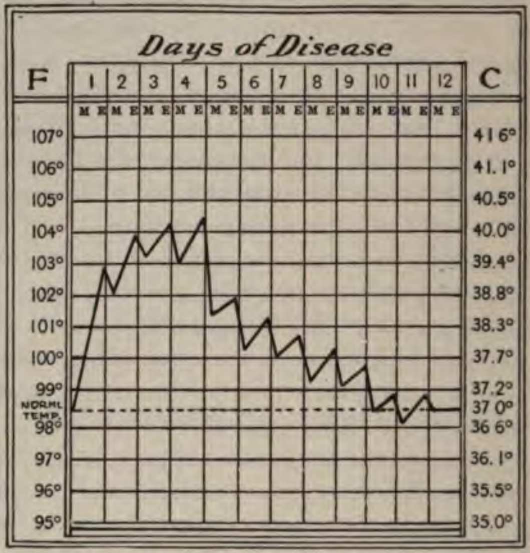
There may be slight delirium even in mild cases during the stage of efflorescence. This delirium may be very active and yet not be of serious import, provided the temperature remains moderate.

The temperature rises when the efflorescence appears, and reaches its maximum at the end of the outbreak, in uncomplicated cases, but there is no decided rise just before or fall after the height of the efflorescence as is the case in measles; on the contrary, the temperature slowly diminishes until the ninth or tenth day from the beginning of the prodromal symptoms, when it becomes about normal, showing no decided crisis such as is seen in measles, but representing a defervescence by lysis. Chart 20 represents the temperature as it commonly occurs in cases of scarlet fever of the benign and regular form.

The pulse is accelerated during the period when the temperature is elevated, and corresponds to it. It varies from 120 to 160.

Desquamation.—The stage of desquamation begins at about the seventh day from the time when the efflorescence first appears, and in the parts of the skin first attacked. The desquamation, however, is not always proportionate to the intensity of the efflorescence. It is at first composed of small particles of cutis, but these soon become larger, and early in the third week from the beginning of the disease they fall from the body in large flakes. This form of desquamation is called *lamellar*. Here, again, we have an important means of distinguishing scarlet fever from measles, for in measles the desquamation is almost universally of a furfuraceous character throughout the whole

CHART 20.



Benign and regular form of scarlet fever.

course of the disease, while the characteristic desquamation of scarlet fever is lamellar. This lamellar form of desquamation may at times, in certain individuals, and following the more intense inflammations of the skin, be represented by large and extensive pieces of skin. This is well shown in a specimen in the Warren Museum, where large strips of skin have come from the hand of a patient with scarlet fever so as almost to form a glove.

Sometimes the desquamation lasts only ten days, but it may continue for two or three weeks. It is especially slow in disappearing from the hands and feet, and it may remain between the fingers and toes for a number of weeks. Sometimes after the desquamation has apparently ceased and the skin has been smooth and normal for several days it may begin again, and thus prolong the period of convalescence.

Urine.—The urine is lessened in amount during the prodromal stage, returns to the normal amount in the stage of efflorescence, increases during the stage of desquamation, amounting at times to a polyuria, and returns again to the normal amount at the end of this stage. During the stage of efflorescence, especially if the temperature is considerably heightened, there may appear in the urine a small amount of albumin, but this disappears as the temperature subsides, is probably only the result of the fever, as in many other diseases accompanied by a high temperature, and is not to be confounded with the albuminuria of the nephritis which in some cases complicates the stage of desquamation.

There is considerable reason to suppose that a mild form of nephritis accompanies almost every case of scarlet fever, although in many cases no clinical symptoms pointing towards the kidney appear and nothing abnormal is found on examination of the urine. This statement, however, rests to such a degree on the authority of the general practitioner, rather than on that of the expert in urinary analysis, that we shall probably in the future find the number of cases which show nothing abnormal in the urine greatly lessened when the number of expert examinations of the urine in mild cases of scarlet fever has increased.

Diagnosis.—The diagnosis of the benign and typical form of scarlet fever is not difficult. Its incubation is decidedly short in comparison with that of any of the other exanthemata. Its prodromal stage is short in comparison with that of variola and measles, and longer than the exceedingly brief prodromal stage of varicella. The characteristic prodromal symptoms of sore throat and a general and intense hyperæmia of the mucous membrane, accompanied by vomiting and severe constitutional symptoms, make it easy to differentiate it from measles, varicella, and variola, none of which, as a rule, show these symptoms.

The punctate erythematous lesions which appear in the stage of efflorescence of scarlet fever are rarely met with in any of the other diseases of this group. This efflorescence, beginning on the neck and chest and extending upward and downward, is distinguished by its peculiar distribution from that of the other members of the group.

The efflorescence which in certain cases appears after the use of antitoxin may simulate so closely the efflorescence of scarlet fever that in the first twenty-four to forty-eight hours it cannot be differentiated excepting by the appearance of the throat. In this connection it must be remembered that the antitoxin efflorescence may appear in forty-eight hours, and again not for three weeks.

The lamellar desquamation is very characteristic, and is seldom seen in any of the other exanthemata. One of the earliest manifestations of the desquamation is a white seam around the base of the nails. (McCollom.)

The complications arising in the ear, and the occurrence of nephritis as a common sequela in scarlet fever, do not to the same degree find their counterparts in varicella and measles.

Prognosis.—The prognosis of the benign and regular form of scarlet fever is in almost every case favorable. It is comparatively rare for the symptoms to become sufficiently serious to cause death unless some complication has arisen in the course of the disease. The individual who succumbs to the simple uncomplicated form of scarlet fever, even when the initial temperature is high and the symptoms are severe, as a rule must have been unusually vulnerable to the toxic effect of the scarlet fever contagium, or must have had a very low degree of vitality at the beginning of the disease.

PROPHYLAXIS.—There is no known prophylactic against scarlet fever except isolation, which for many reasons should be rigorously enforced. We must remember the fact that when the child has passed its tenth year the chances of its ever contracting the disease are very much lessened. We must also appreciate that it is especially important to protect children who are learning, or who have just learned to talk. The commonly occurring and often intractable form of otitis which accompanies scarlet fever may not only render the child deaf, but in a case where the child has not learned to talk it may lead to deaf-mutism. We should, therefore, under all circumstances discountenance the opinion so often expressed by the laity, and sometimes even by physicians, that it is well for children to have scarlet fever while they are young, on the ground that otherwise they will probably contract it at a later period of life, when the type of the disease may be more severe. The assertion that the type of the disease is more severe in adults than in children is not corroborated by my experience.

TREATMENT.—An accurate knowledge of the chief pathological lesions which occur during the course of the disease enables us to deduce the appropriate treatment. By treatment is not to be understood simply the use of drugs. On the contrary, drugs are employed to entirely too great an extent in a large proportion of the uncomplicated cases of the benign type of scarlet fever. I feel that I can speak with some authority on this point, as it has been my rule for many years to compare the results of cases treated by my colleagues with drugs with my own cases treated without drugs, and certainly nothing that I have observed in this comparison would indicate that my patients had suffered from want of treatment. We should have some definite reason for what we do, and should not be influenced by vague ideas of what drugs are supposed to be beneficial in certain diseases.

The treatment of a case of scarlet fever is that of a self-limited disease. With our present knowledge of it, the disease cannot be cut short. We should, therefore, endeavor to keep it within its own limits by avoiding complications. To do this we must remember which tissues are affected as part of the disease and which are likely to be affected by complications.

In the first class we must consider the throat and the skin; in the second class the ear and the kidney.

At the onset of the disease the child, as a rule, is so profoundly affected by the scarlet fever contagium that it wishes to be put to bed at once. The symptoms which from their intensity require treatment in the prodromal stage of the disease are the vomiting, the sore throat, and the high temperature.

The vomiting, as a rule, is of such short duration, and is so symptomatic of nervous gastric disturbance caused by the toxic effect of the poison, that it should be looked upon as eliminative, and usually does not require the use of anything but pieces of cracked ice to be held in the mouth.

The treatment of the throat in scarlet fever is to be especially directed not only to allaying the temporary discomfort of the pharyngitis, but also to the prevention of the inflammatory process from extending through the Eustachian tube to the membrana tympani and producing an otitis which may result in a meningitis. This latter complication is rendered possible by the close vascular connection which exists in infancy and in childhood between the meningeal blood-vessels and the vessels of the tympanum, through the open petro-squamosal suture.

Another reason for systematically treating the throat in all cases of scarlet fever is derived from the belief that the various secondary infections which take place in the disease are probably caused by the entrance of pathogenic organisms to the various tissues through the inflamed and vulnerable mucous membrane of the pharynx. This invasion is commonly of the cervical glands, the ear, the lung, the heart, and the kidney. If this belief is correct, antiseptic treatment directed to the throat is indicated as possibly preventive of the complications which may arise in the disease.

For the purpose not only of allaying the irritation of the throat, but also of preventing the spread of the morbid process to the ear, if possible, the throat and the nose may be sprayed several times during the day. Solutions (two to three per cent.) of borate of sodium in water combined with a small amount of glycerin are useful for this purpose. A four per cent. solution of boric acid in water can also be used to advantage. The local treatment, however, should always be of the mildest form, since any additional irritation of the mucous membrane will render it more vulnerable to the streptococcus invasion. If the child knows how to gargle, the discomfort which arises usually from the sore throat during the first day or two of the disease may often be allayed by simply gargling with cool water. This procedure answers a double purpose: it not only reduces somewhat the hyperæmic condition of the mucous membrane of the upper part of the throat and cleanses the anterior fauces, but also tends to prevent the extension of the pathogenic organisms which would necessarily be favored by a continuous recumbent position of the child. If the child is unable to gargle, some pieces of ice may be given to it to hold in its mouth, and it should occasionally be allowed to sit up, as when its nourishment is being given.

However desirable this treatment of the throat and nose may be in scarlet fever, we are but too often baffled in our attempts to treat them locally, on account of the persistent resistance of the child.

Chlorate of potash, which is so frequently used for the treatment of the throat in scarlet fever, is, in my opinion, a drug which in this disease it will be wiser not to allow the child to swallow, on account of its possible deleterious action on the kidney, which from the beginning of the disease to its end is in so sensitive a condition as to be readily affected by any irritant. Doubtless in a large number of cases we should not be likely to cause renal irritation by the small doses of chlorate of potash which are usually given. Children, however, differ very much in their individual susceptibility to drugs, and we can never tell beforehand whether or not a child is liable to be injured by them. We know that the vegetable salts of potash are decomposed in the system and eliminated as alkaline carbonates, thus causing no irritation in the kidney. Nitrate and chlorate of potash, on the other hand, which do not part with their oxygen in the system, are excreted undecomposed by the kidney, and thus act as irritants. Knowing that the tendency during the whole course of the disease is towards a renal hyperæmia, we should allow the child to have plenty of water to drink.

Unless the child shows decided signs of suffering from a heightened temperature, antipyretics in the form of drugs by the mouth should not be used, as the cases are rare where a temperature of 38.8° to 39.4° C. (102° to 103° F.) for a few days will do harm. This is a safe rule to follow in a disease like scarlet fever, in which, if the child happens to be easily affected by fever, the unfavorable symptoms will appear at once and can be attended to. My opinion is that mere heightening of the temperature without correspondingly severe symptoms causes much needless anxiety. In typical mild cases of the disease, knowing that a lessening of the amount of the urine in the prodromal stage as a result of the high temperature is a part of the regular course of the disease, the administration of diuretics is not indicated beyond a plentiful supply of pure drinking-water. Although the temperature may cause severe initial symptoms, such as convulsions, as a rule it does not have to be directly treated during the prodromal stage. If, however, convulsions occur and continue and the temperature is unusually high, such as 40.5° or 41.1° C. (105° or 106° F.), and if it remains at this height with serious general symptoms, such as delirium, we should endeavor to reduce it by sponging the body with water, the temperature of which should be varied according to the special case. To begin with, the temperature of the water should be about 32.2° C. (90° F.). The child is usually made more comfortable by bathing if the temperature is as high as 40° C. (104° F.). The reduction of temperature by water may be accomplished (1) by sponging, (2) by fanning the child's body wrapped in gauze sprinkled with water of a temperature of 32.2° C. (90° F.), and (3) by placing the child directly in a tub of water and at the same time rubbing the body and limbs and giving a stimulant. The water in the bath should at first be 32.2° C. (90° F.), and gradually reduced five or ten degrees. In extreme cases the cold pack by means of wet sheets can be used; the sheets should be removed in ten or fifteen minutes, the body and limbs rubbed, and a stimulant given.

I have mentioned before that scarlet fever is rare during the first year of life. There are certain observations which seem to show that nephritis is a rare accompaniment of scarlet fever during the first year. We know that milk is the food which is least irritating to the kidney. It would, therefore, seem rational to make milk the diet in a disease which, like scarlet fever, points out to us by its pathology that we should as far as possible avoid irritating the kidney. It may be merely a coincidence, but it seems of some significance that the first year of life should also be the one which is least likely to present cases of scarlatinal nephritis.

When the nausea and vomiting are present, the child, as a rule, feels too sick to take any nourishment whatever. When the violence of the toxic invasion has somewhat abated, and the diagnosis of scarlet fever has been made, orders should at once be given that the child is to have no food but milk. The treatment of scarlet fever with a diet purely of milk has in my practice proved so eminently satisfactory that it has become my routine treatment of the disease. During the initial stage of the disease, and until the stomach has recovered its equilibrium, limewater should be added to the milk in the proportion of one part to ten. Later the alkalinity of the milk can be lessened, and after the early days of the efflorescence the milk may in most cases be given undiluted. As it is possible that the nitrogenous part of the milk may be too great, it is well to reduce the proteids to 2 or 3 per cent. and to increase the sugar to 6 or 7 per cent. The administration of milk alone should be continued through the stages of efflorescence and desquamation, and until we are justified in supposing that a nephritis will not develop in the special case. This in general may be estimated at from four to five weeks from the time of the height of the efflorescence and temperature. Perhaps in this way in a certain number of cases a nephritis may be warded off, and if it develops, the patient is already on a diet which is best suited to the disease.

During the stage of efflorescence there are seldom any symptoms which require special treatment, in the regular form of the disease, except a considerable irritation of the skin which at times arises. This can be allayed by the application either of some simple ointment or of a powder of oxide of zinc and starch (Prescription 62, page 366). The use of the ointment is to be recommended not only because it keeps the skin soft and in good condition, but also because this application reduces the temperature somewhat. Sponging the entire body with water at a temperature of 32.2° C. (90° F.), once or twice daily according to the comfort of the patient, is to be recommended.

During the stage of desquamation the application of a simple ointment to the whole body is desirable both for the purpose of softening the disintegrated epithelium and lessening the duration of this stage, and also to prevent the spread of the contagium by means of the loosened scales.

The child should be kept in bed until the desquamation has almost entirely ceased. This will cover a period of from four to five weeks.

By the end of the fourth week, if the desquamation has completely disappeared, the diet can gradually be increased by the addition of soup and bread. It is well to keep the child in the house for five or six weeks, and still longer if the weather is cold or damp.

The urine should be frequently tested for albumin during the first three weeks, and afterwards, when the child is first allowed to get up, after each change in diet, and after going out. If any albumin is detected, the child should be immediately put back to bed and on a diet of milk until the albumin has disappeared. The mild cases are the very ones in which a nephritis is liable to occur, and therefore we should watch vigilantly until they are out of danger, which is usually in the fifth or sixth week.

Gordon reports a very severe case of scarlet fever with streptococci and staphylococci in the throat, in which rapid improvement occurred after two injections of 10 c.c. of anti-streptococcus serum under the skin. During an epidemic of scarlet fever, in which the mortality was 24.9 per cent., Baginsky treated forty-two cases with Marmorek's anti-streptococcus serum, with a resulting mortality of 14.6 per cent. He, however, points out the variability of the mortality figures in scarlet fever, and for this reason the possible error in attempting to draw any conclusions from the statistics with reference to the serum.

Isolation and Disinfection.—The disease being eminently infectious, the patient with the nurse should be isolated to as great a degree as circumstances will permit. An upper room should preferably be chosen. It has been observed in crowded parts of large cities that scarlet fever in tenement-houses is not so likely to spread when the first cases are in the top rooms of the tenements. In a number of instances in my practice I have had one child of a numerous family strictly isolated in the upper story of the house, and the other children have remained in the house without contracting the disease.

The intensity of the lesions of the skin and the involvement of large surfaces indicate that the air of the room should be kept at an equable temperature, in order that the function of the disabled skin should be taxed as little as possible and that the internal organs should not have too great compensatory work forced upon them. The temperature should be kept at about 20° C. (68° F.).

A disease which renders confinement to the room necessary for weeks demands a room with good ventilation and plentiful sunlight. Therefore a room on the sunny side of the house, having an open fireplace, should be chosen.

The room should be free from all cotton or woollen materials except such as can be destroyed by fire at the end of the disease. The blankets, sheets, towels, and clothes can, of course, be disinfected, but it will save much ultimate trouble to remove the carpet and the curtains and replace them with pieces of old carpet and sheets. The pictures, and, in fact, everything worth preserving, should be removed. The room can be made sufficiently cheerful by means of cheap colored prints and destructible toys to amuse the child.

During the whole course of the disease the greatest care must be taken to disinfect the linen of both the patient and the nurse. This should be done by soaking it for twenty-four hours in a five per cent. solution of carbolic acid, then boiling it for half an hour in water, and finally washing it with soft soap solution, 20 grammes ($\frac{2}{3}$ ounce) to ten litres (10½ quarts) of water.

The dejections are to be received in a vessel one-quarter full of a five per cent. solution of carbolic acid.

After the child is entirely well it is to be thoroughly washed first in a solution of corrosive sublimate 1 to 10,000. The child is then to be taken to another room to be wiped and put into fresh clothes, which have not been in the scarlet fever room. The mattress is to be tied up in canvas wet with a corrosive sublimate solution 1 to 500, and sent out of the house to be disinfected, if possible by steam. I usually advise the family never to have it brought back again. In place of the mattress it is far better to use old blankets, which, if in sufficient number, are comfortable, and at the end of the sickness can be thoroughly boiled and thus disinfected. The useless articles which have been in the room during the sickness should be burned in the open fireplace.

The room must next be disinfected. This is a very difficult matter to do absolutely, but there are several methods which are far better than the usually recommended disinfection by sulphur which has been so generally used for this purpose during the past century. I mention sulphur as a disinfectant merely to state that it was proved by Koch as long ago as 1881 to be entirely unreliable.

If there be paper on the walls, it should be scraped off and immediately burned. The floor should then be washed with a solution of corrosive sublimate 1 to 500, followed by soap water (a mop should be used so as to avoid irritation of the hands). The ceilings, the walls, all the woodwork, and the furniture are to be thoroughly rubbed with bread and then wiped with the corrosive sublimate solution 1 to 500. Esmarch has shown that bread is the best means of removing infectious material from surfaces of this kind. The micro-organisms adhere with great tenacity to the bread, which, with any crumbs that break off and fall to the floor, must be carefully collected and destroyed by fire. The room should then be thoroughly vaporized for eight or ten hours with formaldehyde in the form of pastilles or liquid. For every 3500 cubic feet of space 250 grammes (250 formalin pastilles) of formaldehyde should be used. This can be done with a vaporizing-lamp, the room first having been tightly sealed.

Recent experiments show conclusively that formaldehyde is a reasonably good surface disinfectant, but does not penetrate deeply, and is not so efficient as steam. It is good for walls but not for mattresses or clothes.

It does not appreciably discolor papers or colorings, and does not affect metals.

The room should then be aired for several days. If there are other children in the house, it is well to have the whole room painted, including the ceiling and the floor.

The physician should also bear in mind that the hair, beard, and clothes are possible means of transmitting the contagium from one patient to another, and that it is his manifest duty to the public to change his clothing and disinfect himself on leaving a scarlet fever patient.

The following case illustrates the benign type of scarlet fever without variation from the regular type and without complications:

A boy four and one-half years old was noticed on November 6, when I was vaccinating his sister, an infant, to be quite sick. Besides the infant the boy's two brothers, one two and a half years old and the other six, were in the room with him. The mother supposed that the boy had an attack of indigestion. He had been vomiting quite frequently and had no appetite. His pulse was 120. His temperature was 38.3° C. (101° F.). He had no headache and no sore throat, but he had the appearance somewhat characteristic of scarlet fever well marked on the hard and the soft palate. He was placed in an upper room of the house and completely isolated with a trained nurse. The vomiting continued until evening, when it stopped and did not return.

On November 7 he was reported to have had a restless night. His throat was found to be very much reddened and to feel a little sore. His pulse was 135. His temperature was 38.3° C. (101° F.). He had had a natural movement of the bowels. His appetite was poor.

All unnecessary articles were immediately removed from the room, and he was confined to his bed. He was placed on a diet of milk and given as much water as he wished to drink. The efflorescence of scarlet fever very soon appeared on his chest.

On November 8 the efflorescence had spread all over his body. He was reported to have slept well and to have vomited his milk but once. His pulse was 125, and his temperature was 37.7° C. (100° F.). He was sponged twice daily with water at a temperature of 32.2° C. (90° F.), and as the skin was somewhat irritable the itching was allayed with inunctions of vaseline. The temperature of the room was kept at 20° C. (68° F.).

On November 9 the efflorescence had spread to the limbs, and was also present to a slight degree on the face. At 6 A.M. the pulse was 120, the temperature 36.6° C. (98° F.). At 6 P.M. the temperature was 37.2° (99° F.), and the pulse was 120. He had a little more appetite, his skin was less reddened, and his throat was not so sore.

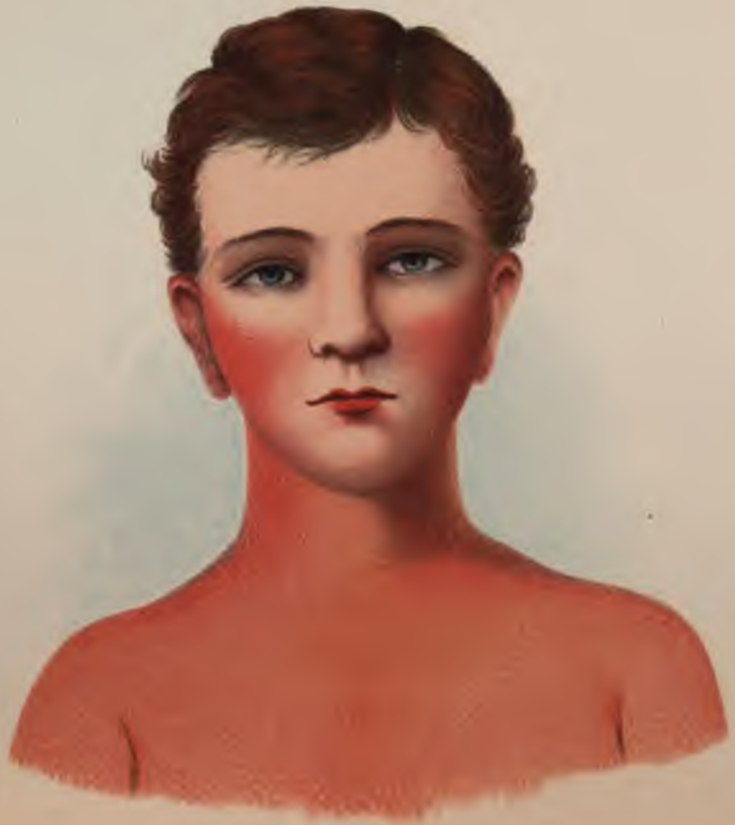
On November 11 the efflorescence began to fade, first on the chest. On November 13 the temperature became normal, and desquamation began, first on the chest. On November 25 the desquamation had almost ceased, and the boy was allowed to get up and play about the room for an hour. On December 1, the desquamation having almost ceased for several days, he began to desquamate freely again. On December 8 the desquamation ceased. He was disinfected and then sent down-stairs among the rest of the children. He went out of doors December 25.

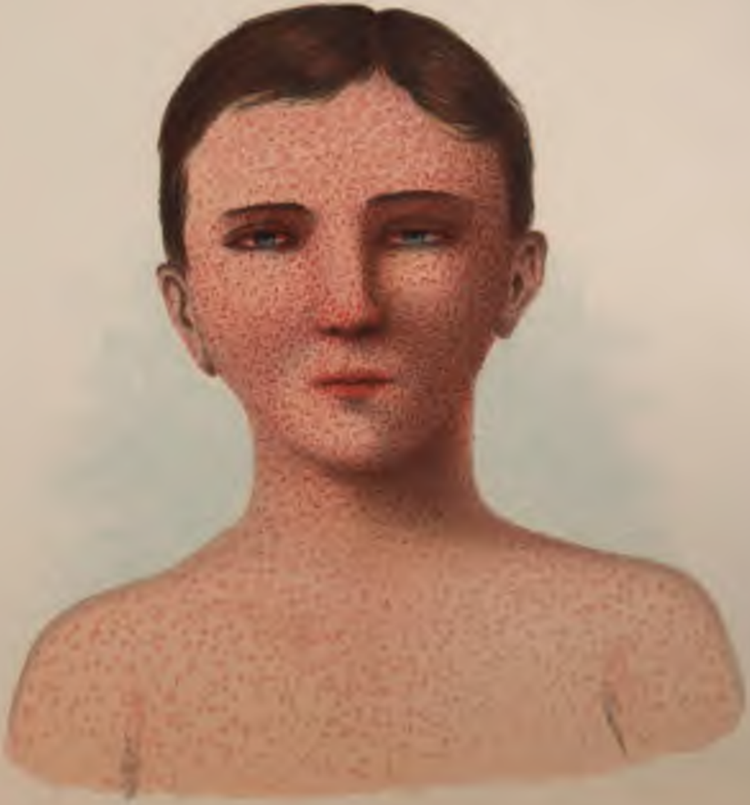
No albumin was detected in his urine during the whole course of the disease. He resumed his usual diet on December 10.

None of the other children contracted the disease, although they remained in the house while their brother was sick.

Plate VIII., facing page 564, is a picture taken from a boy ten years old on the eighth day from the time of infection, the fourth day from the beginning of the prodromal

PLATE VIII.





Measles.

symptoms, and twenty-four hours from the beginning of the efflorescence. The efflorescence was in the form of a punctate erythema.

VARIATIONS IN TYPE OF THE BENIGN FORM OF SCARLET FEVER.—In the benign form of scarlet fever we may have great variations from the typical manifestations of the disease.

A heightened temperature in the evening sometimes continues for over a week after the efflorescence has faded, without the existence of any ascertainable cause: this occurrence should always be looked upon with suspicion. After a rapid increase of temperature at the beginning of the disease there sometimes ensues a condition of complete apyrexia, while all the other symptoms continue to develop in the usual manner. When the temperature remains heightened at the end of the period of efflorescence and continues into the period of desquamation, especially when there is no local pain anywhere, we should suspect that a nephritis may be developing. When the temperature after having become normal rises again, we should suspect such complications as otitis media and suppuration of the subcutaneous tissues of the neck, or that the heart is involved.

Relapses may take place in scarlet fever, but a true relapse is rare, and a second attack, either from auto-infection or reinfection from another individual must be taken into consideration. In the Bagthorpe Fever Hospital, out of two thousand cases fourteen had relapses while under observation. As pointed out by McCollom as a result of his studies in a series of one thousand cases at the Boston City Hospital, we may meet with (1) *pseudo-relapses*, where, after the disappearance of the efflorescence and at the very beginning of the desquamation, symptoms of the disease return; (2) *relapses*, where after the efflorescence has disappeared and during the stage of desquamation the symptoms return; (3) *reinfection*, after desquamation has ceased for a time. The symptoms of these cases are sometimes more severe than those in the first attack, but in most of the reported cases of relapse in scarlet fever the first attack has been a mild one. Such cases occur usually in older children rather than in younger, and must be sharply distinguished from the cases in which a fresh infection has taken place and which are characterized as a second attack of the disease. Thomas reports a case of scarlet fever complicated by varicella, in which on the twenty-fifth day of the scarlet fever a relapse occurred, and on the twenty-sixth day a second attack of varicella developed.

Certain cases of scarlet fever have been reported in which in the latter part of the disease, and after the temperature had become normal, the temperature rose to 40° to 41.1° C. (104° to 106° F.), when no cause could be discovered for the hyperpyrexia, and when the patients recovered after being promptly treated with cold baths to reduce the temperature.

Scarlet fever may begin with such great cerebral excitement as to lead us to suspect meningitis, and it may not be possible to determine the diagnosis until the efflorescence has appeared, which may not be until even the eighth or ninth day.

The efflorescence may last only twenty-four hours, or continue fourteen days. In certain cases a *recrudescence* of the efflorescence may occur. In these cases, which may be showing a high temperature and a brilliant efflorescence, the efflorescence after about forty-eight hours disappears, but the temperature remains elevated, and the efflorescence again appears twenty-four hours later. We must remember that we are not to depend upon the efflorescence in making our diagnosis in scarlet fever, as it may be so evanescent as to be scarcely recognizable. Lemoine, during an epidemic in 1895 of two hundred cases, reports that in thirty-two of these cases the efflorescence was limited to the face. In some extremely mild cases the efflorescence may be entirely absent (*scarlatina sine eruptione*) or evanescent, lasting perhaps only twelve hours. The papillæ of the tongue, however, are enlarged, there is a slight efflorescence on the roof of the mouth, and the diagnosis should, therefore, be made from the mouth. Serious epidemics have been started by this class of cases. While in ordinary types of the disease the efflorescence is not very brilliant, there is another set of cases in which it is very brilliant and is accompanied by a high temperature, which remains elevated for four or five days and then often becomes normal as the desquamation begins.

Convulsions occurring at the onset of the disease are not, as a rule, indicative of a fatal issue, but when they occur later they are usually of serious import.

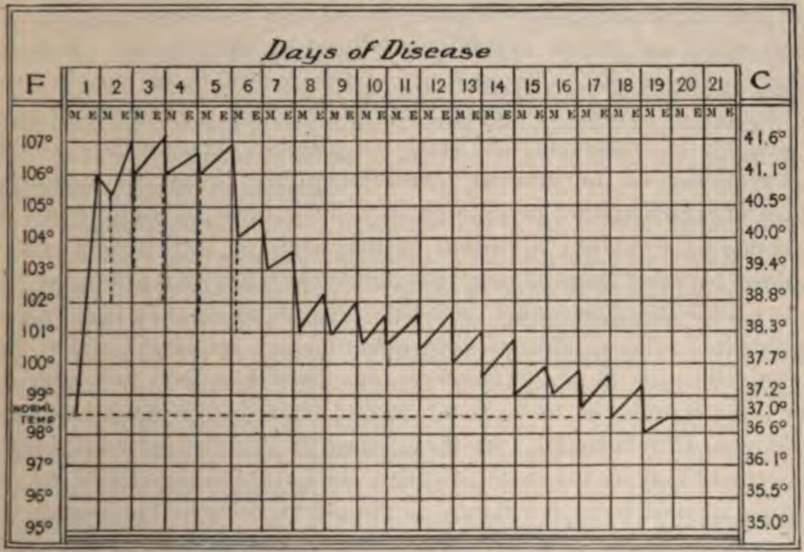
The occurrence of scarlet fever in surgical cases is of no special significance beyond the apparently greater susceptibility of patients with open wounds to contract the disease. We should bear in mind the suggestion of Osler, that in the majority of these surgical cases thus far recorded the efflorescence has probably been the red rash of septicæmia, and that the reported cases have become rare since the gradual disappearance of septicæmia as a complication of surgical operations. Atkinson also suggests that in many instances these rashes may have been due to the quinine which was given to the patient.

A variation may arise from the ordinary scarlatinal inflammation of the mucous membrane of the throat when the efflorescence becomes more severe than usual, resulting in a membranous exudation in the throat and a profuse nasal discharge caused by streptococci and accompanied by very severe symptoms, delirium, and a high temperature for from twelve to fourteen days, the temperature coming down by lysis. The larynx in some cases may also present unusual symptoms, such as aphonia, and serious symptoms caused by a concurrent œdematous condition of the glottis may arise and produce even a fatal issue.

In the benign form of scarlet fever certain cases are at times met with in which the high temperature, or the especial vulnerability of the child to the scarlet fever contagium, causes the symptoms to vary considerably from the typical form and to be unusually grave. An instance of this class of cases was one which was seen by me in consultation with Dr. Robert P. Loring, of Newton Centre.

The child was a girl, six years old. The point of variation from the typical cases of scarlet fever was in this case an unusually high temperature. The invasion of the disease was characterized by restlessness and sore throat, which was soon followed by vomiting and delirium. The temperature on the first day rose to 41.1° C. (106° F.). The highest temperature was on the second and third days, when it reached 41.6° C. (107° F.). During the first three days the pulse could not be counted. The high temperature continued until the sixth day from the beginning of the prodromal symptoms. During the first forty-eight hours there was almost continuous vomiting. This was succeeded on the third day by frequent profuse, and often involuntary, serous discharges from the bowels. These discharges continued until the fifth day. On the fourth day a slight erythematous efflorescence appeared on the neck and chest, and on the fifth day it extended all over the body and was of an intense character. On the sixth day a complication of pain in the wrists began, but it disappeared in twenty-four hours under the administration of salicylic acid. At this time also there was marked swelling on the left side of the neck, which gradually disappeared in four or five days. When the fever was at its height there was considerable cyanosis, with quickened respiration. The pulse at this time was weak and difficult to count. From time to time during the attack antifebrin was given for the restlessness, and bromide of soda was occasionally used. Tincture of digitalis was given when the pulse was rapid and weak and cyanosis was present, but the treatment which was most depended upon was bathing.

CHART 21.



Toxic symptoms and high temperature in scarlet fever treated by baths.

The method of bathing for the purpose of reducing the temperature was that of placing the child in a tub of water. Whenever the temperature reached 40.5° C. (105° F.) the child was placed on a pillow in the bath, and was kept there until the temperature was reduced three or four degrees. The time required to accomplish this tion is simply secondary to a streptococcus invasion, that we are not long in doubt as to our diagnosis, even without the decisive proof by culture. As a rule, when the Klebs-Loeffler bacillus is present the continued increase in the severity of the symptoms and the resulting exhaustion of the child show us that we are dealing with this micro-organism. We must not, however, be misled by this general rule of differential diagnosis, for there are many cases in which it is impossible to differentiate between a streptococcus invasion and an invasion of the Klebs-Loeffler bacillus either by the appearance of the throat or by the clinical symptoms. On the one hand, the streptococcus invasion may be quite as severe in its symptoms as that of the Klebs-Loeffler bacillus, while, on the other hand, true diphtheria may occur where the symptoms are as mild as any that are produced by the other micro-organisms.

Treatment.—The treatment of the throat in these severe secondary conditions is the same as in the treatment of the benign forms, except that, if possible, it should be carried out more rigorously. Although Seitz reports that he has had excellent results from the injection of carbolic acid into the tissues of the tonsils and soft palate, this treatment requires still further proof of its efficacy before it can be recommended. As the disease runs a comparatively short course, there is not such a need for stimulants as is indicated where diphtheria is present. In young children it is often impossible to treat the throat locally, and I have usually found that my chief reliance in tiding over the severe stage of the disease is the administration of sufficient food, and of stimulants when they are indicated by the general condition of the child. It is to be remembered that the throat in scarlet fever may be attacked by the Klebs-Loeffler bacillus and the disease brought to a fatal issue by a complicating diphtheria. When diphtheria is present, the treatment should be the same as for a primary case of diphtheria.

In the more severe forms of inflammation in the throat the inflammatory process may go on to the formation of an abscess, as in the pharynx, but the most common place is in the tonsil or in its neighborhood. These abscesses must be carefully watched for, and when detected opened with antiseptic precautions as soon as possible. We shall by this treatment often shorten the course of the disease, and thus save loss of strength and vitality on the part of the patient.

Cervical Lymph-Nodes.—Symptoms.—The cervical lymph-nodes of the neck are more or less enlarged, according to the severity of the infection. This enlargement may in some cases be so great as to cause much swelling and distortion of the face and neck. The swelling extends at times under the chin from one ear to the other as a mass of cellulitis. The tissues of the neck under these conditions may, as described in speaking of the pathology of the disease, suppurate, and this condition, even if it does not produce a fatal result from gangrene, may greatly prolong the period of convalescence.

TREATMENT.—While the glands are enlarged and tender, the application of hot fomentations usually gives much relief, as does also in some cases an ice poultice. Beyond this I am not in the habit of making any external application.

Ear.—SYMPTOMS.—The middle ear is so closely connected by means of the Eustachian tubes with the naso-pharynx that aural complications are exceedingly common where naso-pharyngeal irritation exists. The symptoms which indicate that a secondary infection of the ear is taking place are not always clear, as they may differ much in their manifestations. We should therefore watch with the greatest solicitude and examine with the greatest care the ear during the course of scarlet fever. The symptoms may be active and represented by aural pain and great restlessness. On the other hand, there may be no apparent pain, especially in infants and young children, who are often unable to indicate the location of the pains by which they are affected. In these cases the symptoms may be merely a somnolent condition and occasional attacks of fretfulness.

TREATMENT.—As soon as an aural complication is detected the treatment of the naso-pharynx should be begun. The nose and naso-pharynx should be kept as clean as possible. The ear should be gently inflated by means of a Politzer bag. Pain should be combated by the instillation into the ear of a solution of atropine in glycerin and water, and by the application of dry warmth. In addition to this, an opiate should, if required, be given internally.

PRESCRIPTION 80.

Metric.	Gramma.	*Apothecary.*	
℞ Atropinæ sulphatis............	0.06	℞ Atropinæ sulphatis.............	gr. i
Glycerini,		Glycerini,	
Aq. destil.....................	āā 3.75	Aq. destil......................	āā ʒi.
M.		M.	

Sig.—Three or four drops to be warmed and dropped into the ear once every three hours.

The congestion should be controlled so far as possible by the internal administration of bromide of potassium in small and frequently repeated doses. If these measures fail to give relief, and if there is an increase of inflammation in the middle ear, as shown by marked swelling and congestion, especially of the superior posterior portion of the membrana tympani, or by a bulging of the membrane, which is seen to be pressed outward by the fluid in the tympanum, paracentesis with the knife should be performed, always with antiseptic precautions and under good illumination. In the early stages of congestion a concentric incision carried along the superior posterior border of the membrana tympani through the congested region, and resulting in free hemorrhage, will often cut short an acute process. A free incision in the most prominent portion of a bulging membrana tympani, by giving a vent to the contained pus, may result in speedy relief from both pain and fever, and justifiably forestall the effort which nature is making to obtain this relief. In the acute congestive stage, after incision of the membrana tympani drainage-wicks made of dry absorbent cotton should be applied, and covered at their outer end with a pad of absorbent cotton filling the concha. These wicks should be renewed as often as both the wick and the cotton pad become saturated. The dressing should be kept strictly aseptic. After the paracentesis of the membrana tympani, in suppurative cases the ear should be syringed frequently with a weak, warm solution of bicarbonate of soda, then carefully dried by means of absorbent cotton, and, after the first few days, dressed by the insufflation of powdered boracic acid, while vaseline may be applied to the canal and concha to guard against excoriation of the skin.

The after-treatment of the middle ear in these cases in which there is no perforation of the membrana tympani should consist in gentle inflation by means of the air-douche used in accordance with the evidence afforded by hearing-tests and by the objective examinations. In cases in which there is perforation of the membrana tympani with continued suppurative discharge, thorough cleansing should be employed. If under this treatment improvement does not soon take place, the patient should be referred to an aurist.

Children are so often rendered deaf by the morbid processes resulting from the scarlet fever contagium that it becomes a positive duty for the attending physician to watch the ear as carefully in these cases as he would watch the heart in a case of rheumatism. In addition to the danger arising from a chronic disturbance of the tissues of the ear, the rapid extension of secondary infection from the naso-pharynx to the middle ear, and thence through the petro-squamosal suture to the cerebral meninges, is a series of complications which usually proves fatal.

The following case illustrates the danger of not treating promptly and thoroughly the complication of otitis media in cases of scarlet fever.

A child two and a half years old had been attacked with scarlet fever and later with a complicating purulent otitis. When I saw the child it was lying in a state of stupor, apparently induced by pressure on the cerebral blood-vessels of an unusually large collection of pus in the middle ear through the petro-squamosal suture. In this case rupture had taken place in both tympanic membranes, and the pus was flowing in large quantities from the external meatus. The perforations of the tympanic membranes were, however, very minute, and the cerebral stupor was not relieved until a free opening was made in each tympanum and the entire middle ear thoroughly syringed out. Although the symptoms of pressure were relieved by these procedures, secondary infection of the cerebral meninges had already taken place, and the boy subsequently died of an acute purulent meningitis.

This case warns us that we should not be misled by the idea that a simple flow of pus from the auricle is necessarily sufficient to provide a proper exit for collections of pus in the middle ear, and that, unless the case is in the hands of an expert aurist, cerebral pressure or purulent meningitis is likely to occur at any time. It also illustrates one of the secondary forms of meningitis.

Kidney.—SYMPTOMS.—It is very important to detect by means of frequent analyses of the urine the beginning of either the milder forms of renal disturbance or of the more severe forms of nephritis, usually represented by that which is called capsular glomerulo-nephritis. If carefully watched for, the appearance of albumin will almost always precede the clinical symptoms, and by a still more rigid enforcement of the rules laid down as practically governing the treatment, the further development of a nephritis may be prevented or at least rendered much less pronounced. It is quite frequently the case that a suspicion is first aroused of the presence of nephritis either by vomiting or by œdema of the face, especially about the eyes, and commonly occurring during the period of desquamation, from the eighteenth to the twenty-fourth day. Under these circumstances the urine will be found to be diminished in quantity and to contain albumin. The daily amount of the urine may be reduced as low as 100 c.c. (3½ ounces), or even lower. The microscopic examination of the urine does not differ materially from that which results from the other forms of nephritis in their early stages, but later we may possibly find that fatty casts are less numerous in the nephritis of scarlet fever, because there is less fatty degeneration in the renal epithelium. The earlier in the course of the disease the symptoms of nephritis appear, the more severe, as a rule, will be its type. The extent of the albuminuria is of less consequence than the total quantity of the urine. A rapid and extensive diminution of the urine is ominous, as it indicates the accumulation of nitrogenous waste in the blood and the danger of a resulting uræmia. The albumin occurring early in the disease is more apt to be in large quantities than when it appears first in the third or fourth week. Hæmaturia is frequently present in this form of nephritis, but ordinarily of itself adds little to the gravity of the disease. The œdema of the face may be followed by a rapid involvement of the ankles and legs and at times may become general. During the course of a general œdema the desquamation is apt to cease, returning on its disappearance. The œdema may last for months or may pass away quickly; it may be entirely absent, but in such cases the nephritis is almost invariably of a light grade.

At times during the presence of a general œdema serous effusions into the pleura may occur. Œdema of the lungs and brain, although rather rare, may also take place. Instead of a slow development beginning with œdema of the face we may have an acute attack, ushered in by fever, vomiting, headache, œdema, amblyopia, coma, and convulsions.

Relapses may occur many weeks after an attack of scarlatinal nephritis, and we should watch the case with the greatest care for several months. Although the nephritis of scarlet fever may last for months, it has a tendency in children ultimately to disappear, on account of their wonderful recuperative powers. It is also rare for the renal disease following scarlet fever to become chronic.

Retinitis and *amaurosis* at times occur during the progress of the nephritis in scarlet fever. In these cases of amaurosis it has been noticed that, although the loss of sight may be complete, almost always when uræmia and amaurosis are coincident there are found no perceptible changes in the retina, no congestion of the papillæ, no increase of intracranial pressure, and no intense œdema of the brain. The sight, under these circumstances, may be recovered completely.

The alterations in the glomeruli already spoken of not only cause the anuria and the uræmia, but also obstruct the renal arteries, as very nearly all the renal blood has to pass through the glomeruli. We find in quite a large number of cases of capsular glomerulo-nephritis a rapid hypertrophy of the left ventricle. This cardiac complication is not to be confounded with the endocarditis which has already been spoken of as secondary to the scarlet fever, and which is supposed to be caused by its special poison or by the streptococci which has already been described as being present in the disease. It is, in fact, not the direct result of the scarlet fever, but is secondary to the nephritis, and is, in this sense, tertiary to the scarlet fever. We therefore do not find this acute cardiac hypertrophy in the earlier stages of scarlet fever, but when a capsular glomerulo-nephritis is once established it may take place in so short a period as a week. This rapid hypertrophy has usually been observed in children between the ages of three and six years, which is of some significance in explaining why this hypertrophy should take place so easily. Between the ages of three and eight years a physiological hypertrophy of the heart exists, possibly caused by a continuance of the aortic narrowing in the neighborhood of the ductus arteriosus, and the heart is more readily affected by increased blood-pressure at that age. This tendency to change in the cardiac muscles is also accentuated by the rapid growth of the organ at this period of life. Besides the cardiac hypertrophy we may, at times, have an acute dilatation of the heart in these cases. This is a serious complication, which must be guarded against, and when it occurs must be recognized at once. These cardiac complications very frequently recover completely, as it is seldom that any extensive changes in the muscles of the heart take place.

Although the occurrence of sugar in the urine during the course of scarlet fever is very rare, yet it is well to examine the urine for this element. By taking this precaution it will sometimes be possible to explain some otherwise obscure symptoms. Zinn reports a case of glycosuria following scarlet fever in a boy four years old who recovered.

TREATMENT.—While very little treatment beyond hygienic measures is needed for the mild uncomplicated cases of scarlet fever, this can hardly be said of the cases that are complicated with severe forms of nephritis, for in these we must act promptly and with great judgment.

We should be careful about using diuretics which might irritate the kidney. Citrate of potash is one of the safer diuretics in this complication. In the lighter cases a lemonade made with bitartrate of potash will be taken well, and will often quickly increase the quantity of the urine, reduce the œdema, diminish the albumin, and cause a radical change for the better. This lemonade may be made by using 4 c.c. (1 drachm) of bitartrate of potash to 473 c.c. (1 pint) of boiling water into which a lemon cut in thin slices has been dropped. This quantity, a little sweetened, may be drunk in twenty-four hours by a child five years old.

In severe cases with general œdema and threatening uræmia cathartics are rather more certain in their action than diaphoretics and diuretics, and are especially indicated when, as is usually the case, constipation is present. Podophyllin in doses of 0.006 gramme ($\frac{1}{10}$ grain) may be given to a child five years old, and repeated a number of times. It usually acts quickly. The compound jalap powder in doses of 0.3 to 0.6 gramme (5 to 10 grains) may also be given when a rapid and decided derivation by the intestine is indicated.

Having provided for the proper movement of the bowels, if the skin is hot and dry, and uræmic symptoms, usually represented by anùria, somnolence, amblyopia, and headache, are present, the hot pack, either wet or dry, should be resorted to. I prefer in these cases to have the child wrapped in a blanket and placed directly in a tub containing water at a temperature of 40.5° to 43.3° C. (105° to 110° F.). The child should be kept in the water fifteen or twenty minutes, and even longer if necessary, and should then be taken from the wet blanket, enveloped in hot, dry blankets and kept in them until the skin has become moist and reaction has taken place. While the child is in the bath, milk can be given to it, and stimulants if they are indicated by a weak or an intermittent pulse.

In addition to this treatment, hydrochlorate of pilocarpine in doses of 0.003 gramme ($\frac{1}{20}$ grain) should be given by the mouth to a child of two years, and subcutaneously, if desired, to a child five years of age. In the cases of threatening uræmia, convulsions sometimes appear quite suddenly. Under these circumstances enemata of hydrate of chloral, 0.3 to 0.6 gramme (5 to 10 grains) dissolved in water, are of value in controlling the nervous phenomena. A combination of bromide of potash and hydrate of chloral can also be used as in the following prescription :

PRESCRIPTION 81.

Metric.	Gramma.	*Apothecary.*	
℞ Chloral hydrat.	7.5	℞ Chloral hydrat.	ʒii ;
Potassii brom.	15.0	Potassii brom.	ʒiv ;
Aq. destil.	90.0	Aq. destil.	℥iii.
M.		M.	

Sig.—3.75 c.c. (1 drachm) in 30 c.c. (1 ounce) of warm water: to be given by enema, and repeated in half an hour if needed.

Where the ascites is extreme, paracentesis abdominis is often of great value, not only in relieving the pressure, but also in increasing the action of the diuretic, which, perhaps, before was not acting freely. Digitalis is a valuable remedy especially adapted to the treatment of the nephritis of scarlet fever and to that of the cardiac changes which result from it. By the administration of this drug the flow of urine is increased. It is best given in the form of a freshly prepared infusion, in teaspoonful doses every four hours to a child five years old. Diuretin, 0.3 gramme (5 grains), dissolved in water and given two or three times in the twenty-four hours, has proved of considerable value in mv cases, and is apparently harmless.

Joints.—SYMPTOMS.—An acute inflammation of the joints, usually the larger ones, is not infrequently met with during the course of scarlet fever. It is more frequent in adults than in children, and usually occurs at the end of the first week (Marsden). This acute synovitis is at times apparently either due to or closely connected with rheumatism, and may be accompanied by endocarditis and pericarditis. The latter disease is, however, rarely met with unless in the later stages of scarlet fever in cases in which nephritis has developed. These rheumatic cases are usually controlled by the administration of salicylic acid. As a rule, they are not of long duration, and if effusion takes place in the joints it is serous, does not become purulent, and does not give an especially serious prognosis. True rheumatic synovitis in convalescence is very rare.

In connection with these cases, when uncomplicated or when the heart is also affected, chorea has sometimes arisen as a complication.

A more severe form of synovitis, apparently caused by sepsis, may also occur during the course of scarlet fever. The effusion in the joints in these cases may become purulent and lead to serious and permanent disorganization of the tissues and often to death from general septic infection.

Besides these acute inflammations of the joints a chronic process at times arises, appearing, as a rule, very late in the disease or subsequent to it by many months. This inflammation is tubercular in character, and affects with especial frequency the hip and knee. Although tubercular, it seems to be a late result of the original toxic effect of the micro-organisms of or secondarily connected with the scarlet fever contagium.

In addition to these more common complications of scarlet fever a number of secondary infections are at times met with. Thus, cases of purpura following or complicating scarlet fever have been reported. and are usually fatal.

Bruck reports three cases of myositis of the large muscles following scarlet fever, the symptoms being pain, tenderness, and increase in the size of the muscles.

The following case represents one of the milder forms of what was probably capsular glomerulo-nephritis, and the effect of rest in the treatment of the disease.

A girl, five years old, was attacked by scarlet fever of the benign form and very mild in its character. After the usual prodromal symptoms the efflorescence appeared and ran its course, and desquamation became established. At the end of the second week, and while the desquamation was still present, the child seemed so well that it was allowed to be dressed and about its room. It was also allowed to have its usual food, which included a considerable amount of meat.

On January 4 the child was very irritable during the day and passed her urine involuntarily in the forenoon. During the afternoon she was feverish, and passed frequently small amounts of urine. That night she slept well, but on awakening on the morning of January 5 she seemed dull, and was said to be feverish and to have little appetite.

On January 6 the record stated that she had passed only 90 c.c. (3 ounces) of urine in the twenty-four hours. She seemed tired and languid, and there was an œdematous condition of the eyes and upper part of the face. She had one normal movement of the bowels.

On January 7 the total amount of urine passed in the twenty-four hours was 480 c.c. (16 ounces). She was given infusion of digitalis and cream of tartar water on this day, and placed on a diet of milk.

On January 8 she seemed better, and passed 480 c.c (16 ounces) of urine in the twenty-four hours. She was then allowed to have an increase in her diet, consisting of various kinds of broth. An examination of the urine by Professor E. S. Wood on this day gave the following result :

Color	Rather pale.
Reaction	Acid.
Urophæin	Diminished.
Indoxyl	Increased.
Urea	Diminished.
Uric acid	Increased.
Albumin	Considerable trace.
Sugar	Absent.
Bile-pigments	Absent.
Specific gravity	1009.
Chlorides	Almost absent.
Earthy phosphates	Diminished.
Alkaline phosphates	Diminished.
Sediment	Slight in amount; consisted chiefly of normal blood-globules, a few renal cells, and a few hyaline, fibrinous, blood, and epithelial casts. The blood-globules and the casts were normal in appearance.

In regard to this examination Professor Wood remarks that the important features of the urine were its dilution, the great diminution in the normal salts, especially in the chlorides, the considerable trace of albumin, and the blood and casts. The normal character of the blood-globules and the comparatively small number of the casts seemed to show that only a small portion of the kidney was affected. At the time of the great diminution in the quantity of the urine the tubules were probably nearly completely blocked up. The low specific gravity and the great diminution of the urea and chlorides seemed to indicate that it would need but little additional irritation to produce a marked nephritis. The condition appeared to be one of a mild nephritis.

The general symptoms presented by the child and the disturbance of the kidney shown by the examination of the urine made me advise that she should be kept in bed in a warm room and placed on a diet exclusively of milk. A warm bath was to be given once or twice daily until a larger amount of urine was passed, and 4 c.c. (1 drachm) of infusion of digitalis administered four times in the twenty-four hours.

On January 9 the total amount of urine passed in the twenty-four hours was reduced to 90 c.c. (3 ounces), and the child was nauseated and vomited a number of times during the day.

On January 10 she was reported to have had a very restless night and to have been very much excited on awakening. She had no pain anywhere. Her face continued to be œdematous. The total amount of urine passed in the twenty-four hours was 240 c.c. (8 ounces). She perspired slightly, and had one large, loose dejection. She so absolutely refused to take milk that she was given 103 c.c. (3½ ounces) of beef-juice, which was all the nourishment that she took on this day.

On January 11 the face was more œdematous, and she was languid. She had two large, loose, offensive dejections from the bowels, and complained of a burning sensation in the rectum at the time of the movements. The total quantity of urine was 300 c.c. (10 ounces). On this day she was finally persuaded to take milk, and no other food was given to her.

On January 12 the child seemed brighter and the face was not so much swollen. The total amount of urine in the twenty-four hours increased to 540 c.c. (18 ounces). Complete recovery took place in three and one-half months from the beginning of the attack.

During the course of her sickness various attempts were made to increase her diet more quickly and to allow her to be dressed and about the room, but each time when this was done she showed symptoms which pointed towards the presence of a renal complication, such as a swelling of the eyes and face and a rise of temperature, with resulting nausea and loss of appetite.

This case shows how careful we must be for many weeks and even months to control the temperature of the room, the amount of exercise, and the kind of food, when nephritis has complicated a case of scarlet fever. It also shows how entire recovery may take place even when the renal irritation is pronounced and unusually prolonged.

Fig. 182, on page 855, represents a case of scarlet fever complicated by a probable capsular glomerulo-nephritis and a resulting cardiac enlargement. The following is a record of the case:

A boy, seven years old, entered the hospital on July 28. His mother was living and well, and stated that his father died of Bright's disease. The child was said to have been well until five and a half years old, when he had an attack of scarlet fever, mild in form and not accompanied by any severe symptoms. In the latter part of the attack his temperature rose and he began to have dyspnœa and dropsy. Since that time he had been slowly but steadily growing worse. He had extensive œdema of the face, chest, arms, abdomen, and legs. He was somewhat cyanotic, and his breathing was so much affected that he was unable to lie down, the orthopnœa compelling him to be supported in a semi-recumbent position. There were a slight puffiness about both eyes and a yellow tinge of the conjunctivæ. The lips and tongue were cyanotic. The extremities were cold to the touch, and the skin pitted readily on pressure. The skin of the whole body was dry and harsh and in certain portions covered with fine scales. On the inner side of the left leg and on the outer side of the right leg were some old scars, apparently resulting from a previous scarification performed for the reduction of the anasarca. In addition to the œdematous condition of the walls of the abdomen, a distinct fluctuation was found on palpation. An examination of the lungs showed that there was dulness over both bases behind, and over these areas of dulness, as well as over the whole front of the chest, fine moist râles could be heard, indicating an œdematous condition of the lungs. The heart's impulse was most distinct in the sixth interspace a little outside of the mammillary line. The area of cardiac dulness extended from the second rib on the left to 2.5 cm. (1 inch) to the right of the sternum, in an area corresponding to the third interspace and fourth rib. The dulness then extended to the left across the sternum to a point 2.5 cm. (1 inch) outside of the mammillary line and as low as the sixth interspace, corresponding to the cardiac impulse. A loud systolic murmur could be heard over the region of the cardiac impulse, and was transmitted so that it could be heard in every part of the thorax. The total amount of urine in twenty-four hours varied from 900 to 1050 c.c. (30 to 35 ounces). An analysis of the urine gave the following results :

Color	Darker than normal.
Specific gravity	1013.
Reaction	Acid.
Urophæin	Diminished.
Indican	Increased.
Chlorides	Diminished.
Albumin	⅔ per cent.
Sugar	Absent.
Sediment	Very slight and flocculent. Microscopic examination showed numerous short hyaline and granular casts of medium diameter and occasionally of small diameter ; an excess of renal epithelium ; considerable abnormal blood ; an occasional white corpuscle ; one or two blood-casts, many hyaline and granular casts, with one or m renal cells adherent ; occasional fatty renal cells and casts with few fat-drops adherent.

July 29 he was unable to lie down with comfort, on account of dyspnœa arisin from an accumulation of fluid in the abdomen. The legs were also very much swolle and œdematous. His face was somewhat puffy. The cyanosis was marked and th child had considerable dyspnœa.

Paracentesis of the abdomen was performed, and after 480 c.c. (16 ounces) of clear, yellowish fluid were removed the cyanosis and dyspnœa were decidedly diminished.

The treatment was absolute rest, so as not to tax the muscles of the heart more than possible, hot baths to increase the action of the skin, laxatives to relieve the congested condition of the kidneys, and non-irritating diuretics, such as citrate of potash and digitalis.

For the next few days after paracentesis of the abdomen the child improved greatly, the dyspnœa ceased, the urine became of a better color and increased in amount, the cyanosis grew less, and, although the pulse was still small and feeble, the child showed great general improvement. In the course of a month the œdema was so much reduced that the child looked like a different person. He was able to lie down with comfort, slept well, his appetite returned, and at one time he could even be moved about the ward in a wheel-chair. Some weeks later the cardiac symptoms returned, and he again began to have œdema and ascites, cyanosis and orthopnœa. The symptoms were mostly those of a crippled heart.

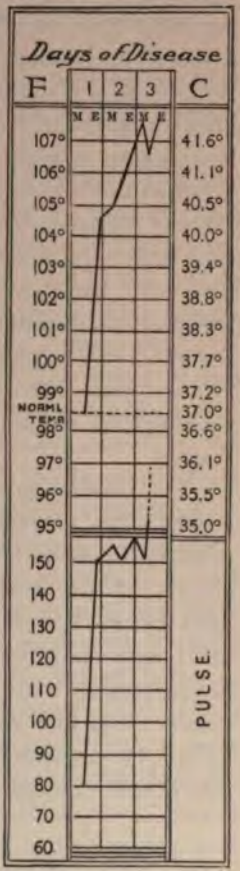
On September 8 the œdema increased, and the urine was reduced to 450 c.c. (15 ounces). Diuretin was given in doses of 0.6 gramme (10 grains), which increased the flow of urine to 1230 c.c. (41 ounces). The diuretin given in these doses once or twice a day for some time continued to act successfully.

In October the action of the heart grew still weaker, the œdema of the lungs increased, and, although there had been a general improvement, the child grew progressively weaker during November. Early in December he was attacked with vomiting, had a weak and rapid pulse, gradually failed in strength, and on the 21st of December died suddenly. No autopsy was obtained.

MALIGNANT FORM OF SCARLET FEVER.

The malignant form of scarlet fever is almost without exception fatal, and is very rare in comparison with the benign form. Malignant scarlet fever appears to attack those individuals who have a predisposition to be profoundly affected by the scarlet fever contagium. In these cases we see healthy children attacked with intense headache, high fever, delirium, sometimes coma, and death follows usually in two or three days, the course of the disease being unaffected by treatment. In these cases, however, the anti-streptococcus serum should be used, as it has not yet been proved that it may not arrest the disease. A case of this kind was seen by me in consultation with Dr. Emerson, of Concord, and represents the conditions which are present in these cases of malignant scarlet fever.

CHART 22.



Malignant form of scarlet fever. Girl, 11 years old.

A girl, eleven years old, was perfectly well and strong and had no other diseases up to January 10. In the middle of the day she felt very ill and vomited. Her pulse was 150, temperature 40.2° C. (104.5° F.). The pharynx and tonsils were much reddened, but there was no exudation or membrane to be seen. An efflorescence of a scarlatinal type appeared on the chest in the afternoon. The vomiting continued through the night and up to the morning of January 11. The child was conscious, but dull. The pulse was 150, and the temperature was 40.5° C. (105° F.). At 4 P.M. the face became puffy, and the efflorescence was well marked on the body and extended to the extremities. The child was wandering and stupid, and the temperature rose to 42.2° C. (108° F.). The extremities became livid, and the vomiting began again. At 6.30 P.M. the temperature, after the internal administration of various remedies, was found to be 41.6° C. (107° F.), and at 10 P.M. 41.1° C. (106° F.), and the pulse 160, weak and difficult to count. At 6 A.M. on the 12th, within forty-eight hours from the appearance of the first symptoms, the child died

The case was a perfectly hopeless one from the beginning, as every method of treatment which could be thought of was tried and proved absolutely fruitless. Tub bathing with water at different temperatures, and finally sponging with ice-water, had no effect whatever on the temperature or the general symptoms.

Chart 22 shows the temperature from the time of the attack to within a few hours before death.

MEASLES.

Measles (rubeola) is an acute infectious disease, supposed to be caused by a specific micro-organism, and characterized by lachrymation, photophobia, coryza, cough, a papular efflorescence, and a slight desquamation.

ETIOLOGY.—The micro-organism which produces measles has not yet been determined. It is supposed to find its vehicle in the tears, and in the secretion of the throat and nose, and possibly to exist in the blood. Its tenacity for clothing, thus continuing as a fresh source of infection, is mild in comparison with that of scarlet fever. It is very infectious, and in some communities is at times exceeding fatal. This was the case in the epidemic of 1873 in the Fiji Islands, where it had not occurred for a long time; it spread rapidly, and caused two thousand deaths, of which sixty-seven per cent. were in children under five years of age. The high mortality in measles is, as a rule, not caused by the measles itself, but by its complications. The epidemics of measles spread rapidly and appear to have an element of periodicity. This has been well exemplified in Boston, in the crowded districts at the North End, where in certain years large numbers of children are affected, and where in the succeeding years the disease appears only sporadically. Measles can occur three or four times in the same individual: this recurrence was one of the peculiar features of the epidemic in Boston in 1880. It may attack young infants, but is rare under six months. After the sixth month, and especially during the first year, the susceptibility to the disease is increased, and we meet with the greatest number of cases between the first and the fifth year. The susceptibility to measles appears to lessen as puberty is approached. It is somewhat rare in adult life, although the fact of its attacking large numbers of adults was also a peculiarity of the epidemic of 1880 in Boston.

The contagium apparently passes from one individual to another after a very short exposure, and often without any direct contact, as by transmission through clothing or by the hands. It is most infectious in the beginning of the attack, and the infection may be transmitted three or four days before the efflorescence appears on the skin. There seems to be much less liability for the transmission of the disease during the stage of desquamation than is the case in some of the other exanthemata, such as scarlet fever and variola, the means of transmission corresponding more to that of varicella. The following case illustrates the high degree of the infection in the early stages of measles.

A boy who was in my ward at the Children's Hospital was attacked with scarlet fever. I had him removed to the contagious ward and placed under the care of a special nurse, who had orders to carry out the most precise antiseptic treatment. The directions to the nurse were that she should apply an ointment to the child, rubbing it into the skin thoroughly from the head to the feet twice daily. The child was also to be bathed twice daily with a solution of corrosive sublimate, 1 to 10,000. The nurse was cautioned not to allow her clothes to touch the boy's bed.

During the early stage of this boy's desquamation a second boy, who occupied the bed in the general ward next to the bed from which the first boy had been removed, was attacked with sore throat, vomiting, and fever. I had already paid my visit for the day, and my house officer, thinking the case was probably one of scarlet fever contracted from having been in such close proximity to the bed from which the first boy was taken, had the second boy removed to the contagious ward and placed in the same room with the first boy. On the following morning I found that the second boy did not have scarlet fever, but had measles. I immediately had the second boy removed to another room, and he was carefully watched for a week, supposing that having passed the night with the first boy, who was in the most infectious stage of scarlet fever, he might have contracted scarlet fever. A week passed, and he evidently had escaped infection by the scarlet fever contagium.

Ten days later the boy who had scarlet fever was attacked with measles, presumably contracted during the night from the boy who was his room-mate in the early stage of his attack of measles.

These two cases apparently show—first, that scarlet fever, even during its most infectious stage, can in some cases be prevented from spreading by thorough and constant disinfection; secondly, that measles is highly contagious in its early stages.

Pathology.—Beyond the morbid conditions which appear on the *skin* and on the mucous membrane of the throat, there is no especially characteristic pathology of measles.

Neumann has studied the pathology of the skin in measles by means of specimens which were hardened in a dilute solution of chromic acid and colored with carmine, hæmatoxylin, and picro-carmine. The pathological changes were found to be almost entirely confined to the glands of the skin and to the blood-vessels. About the walls of the blood-vessels, principally in the upper layers of the cutis, were found collections of round cells which in crowded masses surrounded the loops of the blood-vessels even in the papillæ. The blood-vessels themselves were dilated and full of blood. The coils of the sweat-glands, as well as the excretory ducts, were enveloped in accumulations of round cells, while the neighboring tissues were filled with these cells. These collections of cells were always situated outside of the walls of the glands. The sebaceous glands presented like changes. The hair-follicles showed rounded protuberances which corresponded to the points of insertion of the erectores pilorum, and which were probably caused by contraction of these muscles. In the muscles themselves there were to be found, between the cells proper of the muscular tissue, scattered round cells, which showed the participation of the muscular tissue in the inflammatory process. The hair-follicles, in the same manner as the sweat-glands, were seen to be surrounded in their entire length by collections of round cells, which were more numerous in the lower than in the upper part of the skin. We therefore see that in measles the pathological process in the skin affects chiefly the blood-vessels and glands, while the tissue proper of the skin, as well as of the epithelium, presents no marked changes.

From the fact that in measles the pathological processes of the disease are situated more particularly around the blood-vessels and cutaneous glands, it may be assumed that the infectious material of the malady, whatever its nature, is eliminated from the system through these channels.

In addition to the pathological lesions which occur in the uncomplicated cases of measles, there is almost always associated with the catarrhal condition of the mucous membrane of the upper air-passages a catarrh of the larger bronchi. One of the most common complications of measles is pneumonia; this is usually a broncho-pneumonia, lobar pneumonia being comparatively rare.

In some cases an inflammation of the smaller bronchi accompanied by pulmonary collapse occurs. The bronchial glands are apt to be swollen if the secondary infection is a severe one. According to Osler, a swelling of Peyer's glands is not uncommon, and may be accompanied by a hyperæmic condition of the mucous membrane of the gastro-enteric tract.

Although a secondary infection of the ear has been considered rather distinctive of scarlet fever, this complication has in my experience arisen also quite frequently in measles. When the ear is affected in measles there is a congestion of the middle ear. When the onset of the preliminary congestion occurs in connection with the inflammation of the nasal and naso-pharyngeal mucous membrane, it consists of a simple, general, acute congestion of the middle ear, accompanied in the beginning with serous exudation, and later with a rapid thickening of the membrana tympani in connection with the inception of the suppurative process. When, on the other hand, the preliminary congestion is coincident with or follows the efflorescence on the face, the congestion is primarily in the upper portions of the membrana tympani as the result of a suspension of vasomotor inhibition. Under these conditions there is a congestion of the manubrial plexus, of the superior and posterior portions of the membrana tympani, and of the corresponding portions of the inner end of the external auditory canals.

In addition to this more common condition, a general congestion of the membrana tympani is found during the stage of efflorescence, and is likely to be more severe in its type than that which occurs during the prodromal stage of measles.

The inflammation of the middle ear accompanying measles is more likely than is scarlet fever to leave behind such trophic changes as thickening of the tympanic mucous membrane with the formation of adhesions.

During an attack of measles, and subsequent to it, the tissues show an especial vulnerability to infection by the tubercle bacillus. The tubercular infection may be represented by the lesions of a general miliary tuberculosis or by those of especial tissues, such as of the cervical and bronchial glands, the joints, the ear, and, most commonly of all, the lung. In the latter instance the lesions are usually those of a tubercular broncho-pneumonia.

INCUBATION.—The time of the incubation of measles may vary very much, and may cover a period of two or three weeks; the usual time, however, is ten days.

SYMPTOMS.—*Prodromata.*—The prodromal stage varies in length, but, reckoning ten days as the usual time for the stage of incubation, the prodromal stage may be considered to last from two to three days, and in some cases four days. In this stage we have in typical cases of the disease symptoms distinctive of measles. The invasion is characterized by severe catarrhal conditions affecting the nose (coryza), the eye (lachrymation), and the throat and upper air-passages (cough). In the first twenty-four hours the temperature rises to 38° or 39° C. (100.4° or 102.2° F.), and often to

40° C. (104° F.). The height of the temperature on the first evening is a fair indication as to the severity of the coming disease. Thus, a temperature of 40.5° C. (105° F.) indicates a severe case. An important point to be noticed regarding the prodromal symptoms is that after the first twenty-four hours there is in a large number of cases a remission in the temperature, which goes down, perhaps, to 37.5° or 37° C. (99.5° or 98.6° F.), and remains down for about twenty-four hours, when it again rises. The cough, coryza, and lachrymation, which appear early in the prodromal stage, do not abate, but rather increase, during this remission of the temperature. This is an important point to remember, as the child who seems quite sick and loses its appetite while the temperature is high during the invasion of the disease, seems brighter and has a return of appetite on the second day when the temperature is lower. This peculiarity of the prodromal stage is often misleading both to the parents and to the physician, who, because the child appears so much better, are led to believe that one of the infectious diseases is not developing. In infants and young children the prodromal stage may begin with a convulsion, but this is unusual, and if it occurs it is not, as a rule, particularly severe, and does not necessarily make the prognosis more grave. Headache in the prodromal stage is quite frequent; vomiting is rather rare. The tongue is usually furred, and the mucous membrane of the throat towards the end of the second day, and before the efflorescence has appeared on the skin, shows a condition which is very similar to that which is about to appear on the skin. These lesions, which are especially pronounced on the soft and the hard palate, are represented by papules or macules of a dark-red and later purplish-red color, of different sizes, and considerably larger than the punctate macules which were described in connection with the throat in scarlet fever. These papules in certain cases are arranged crescentically, and may sometimes be found to have coalesced in some parts of the fauces. The mucous membrane between the lesions is comparatively normal in color, although there may be a slight hyperæmia of the entire throat. This hyperæmia, however, is not nearly so intense as is seen in the throat in scarlet fever. Forchheimer states that the exanthem in measles begins upon the soft palate from thirty-six to forty-eight hours before the efflorescence, in the form of purplish or bluish papules arranged crescentically, extends over the mucous membrane of the cheeks, and is accompanied by a bluish color of the tongue. The exanthem is at its maximum with the beginning of the efflorescence, and may take as long as three or four days to disappear.

In addition to the efflorescence in the throat, Koplik has described certain spots on the mucous membrane of the cheeks which appear during the prodromal stage. He describes these spots as minute bluish-white specks in the centre of a reddish areola. Widowitch has found them in 86.6 per cent. of cases of measles, in 8 per cent. of cases of rubella, and in less than 1 per cent. of other cases.

After the remission of the temperature, on the second day, the temperature again rises on the third or fourth day.

Efflorescence.—At the end of the third day or at the beginning of the fourth day—that is, the thirteenth or fourteenth day from the time when infection took place—an efflorescence appears on the skin. The efflorescence usually reaches its maximum in about thirty-six hours, this being a more constant number than the other figures; that is, it is about the fifteenth day from the date of infection. The stage of incubation is rather more constant than the stages of prodrome and efflorescence, the latter two varying as to their length, but together amounting to five or six days.

When the efflorescence appears on the skin it consists commonly of small macules or papules on a slightly reddened base, which first appear on the face. As the disease progresses, these lesions extend to the neck and chest, and in the latter locality are, especially in the beginning, of a delicate pink color, the form of distribution in some cases being crescentic. The efflorescence then rapidly extends to the rest of the body and to the extremities. It is usually more pronounced on the face, where the papules are apt to coalesce, and where an œdematous condition of the tissues, especially around the eyes and nose, usually occurs. The eyes are swollen and partially closed, and the conjunctivæ are reddened. Photophobia at this time is pronounced. The efflorescence may also appear on the scalp. It remains well marked for from one to two days, and while it is at its height the temperature reaches its maximum, and remains high for two or three days, corresponding to the intensity of the efflorescence. It then rapidly falls, and reaches the normal point in about two days more,—that is, there often appears to be a distinct crisis in the disease. During the period of efflorescence, when the temperature is still raised and the efflorescence is at its maximum, it is usual to have, in addition to the symptoms of cough, coryza, and lachrymation, a slight disturbance of the intestines, represented by small, frequent, loose discharges, apparently arising from irritation of the rectum and descending colon. This condition is seldom a serious one, and no especial attention need be paid to it unless it should continue for some days, or after the maximum of the temperature and efflorescence has been passed for a day or two.

DESQUAMATION.—The desquamation is usually furfuraceous in character,—that is, the epithelium is cast off in fine flakes, and is thus distinguished from the large lamellar flakes occurring during the period of desquamation in scarlet fever. The desquamation begins in the order in which the efflorescence came out,—namely, first on the face and later on the chest. The furfuraceous character of the desquamation is especially noticeable on the sides of the nose. The disease usually runs its entire course in three weeks.

DIAGNOSIS.—In order to understand how difficult it sometimes is to diagnosticate measles, we must recognize that it is one of the most variable diseases with which we have to deal. During epidemics of undoubted measles cases arise which differ materially from the disease as it appears in its typical form, yet these cases, by producing the typical form in other individuals, prove that they are all caused by the same contagium. In like manner certain epidemics may be characterized by irregular forms of the disease, and, as true measles can occur a number of times in the same individual, the recognition of a sporadic case is often impossible. As in other diseases of the skin, we should recognize measles not by any particular dermal lesion, but by the peculiarities of the prodromal symptoms, the general course and location of the efflorescence, the time of the maximum of the efflorescence and temperature, and the character of the desquamation. Thus, a prodromal stage of three or four days, characterized by catarrhal symptoms of the eyes, nose, and upper air-passages, by the presence of Koplik's spots in a large majority of cases, and a papular efflorescence appearing first on the face, differentiates the disease from variola, varicella, and scarlet fever.

Prognosis.—The prognosis of measles, as a rule, is good, but this depends almost entirely upon whether the disease is free from or accompanied by complications.

Treatment.—The treatment of measles is essentially symptomatic. There is no known means of producing immunity to the disease or of shortening its course. It is a self-limited disease, and the treatment should be directed towards the protection of the organs which are most likely to be attacked by complications. Bearing in mind that the eye, the nose, and the throat are affected in the prodromal stage, that later the skin is in a very sensitive condition, and that the lung is frequently the seat of some complication, we should direct our treatment especially to the care of these organs.

The child should be placed in a room kept at an equable temperature, 20° to 21.1° C. (68° to 70° F.), and well ventilated. The room should be darkened, and the eyes should be protected from light during the whole course of the disease. Unless this precaution is taken, the eyes are often seriously affected for many months after the measles itself has disappeared. The child should be kept in bed until the temperature has been normal for a few days, the efflorescence has faded entirely, and the desquamation has almost ceased.

The diet during the period of the height of the temperature should be soup, milk, and bread. Later, when the temperature is normal and desquamation has begun, the child can gradually have its diet increased, until by the third week from the beginning of the attack it is having its usual food.

The cough, which is very troublesome at times, does not, as a rule, require any special treatment, as it will of itself in most cases pass off in a few days. While it continues it can be treated with some simple mixture, such as camphorated tincture of opium in cold water in doses of 0.3 to 0.6 c.c. (5 to 10 minims), to allay the irritation in the throat.

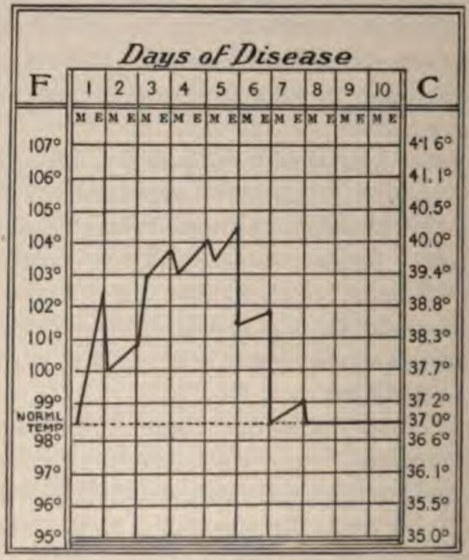
For the irritation of the nose, atomizing the nares with some simple refined oil, such as oleum petrolatum album, is useful. During the invasion of the disease, however, these catarrhal symptoms are exceedingly difficult to control by any treatment whatever.

As at times there is great irritation of the skin during the period of efflorescence, a powder (such as Prescription 62, page 366) should be applied thickly to the entire body and limbs. In place of the powder some simple ointment, such as petrolatum, may prove to be more soothing.

As a rule, the child should be kept in an equable temperature for at least three weeks, and at the end of that time, if the desquamation has ceased, it may be allowed to go out of its room, and out of the house a few days later in pleasant weather. For several months, however, it should be carefully protected from sudden changes of atmosphere, as the catarrh of the air-passages is so likely to leave them in an extremely sensitive condition that a very slight irritation may cause a recurrence.

Before the child is allowed to leave its room it should be thoroughly bathed from head to foot in hot water. Although the contagium of measles has not the same tenacity for clothing as the contagia of variola and scarlet fever, yet the room should be thoroughly disinfected after the child has left it. This can be done in the same way as described in speaking of scarlet fever; but the extreme precautions taken in the latter disease are not considered necessary for the prevention of the extension of measles. If the carpet had not been removed when the child was put into the room, it can be taken from the house and thoroughly cleansed before it is brought back. The bedclothes and

CHART 23.



Typical measles.

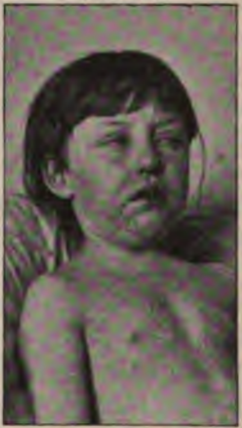
everything that can be washed should be thoroughly boiled. The room should be cleansed and the windows allowed to remain open for several days, as fresh air is one of the best means of eradicating the micro-organisms connected with the exanthemata.

Chart 23, page 586, shows the temperature as it usually occurs in the typical and regular form of measles.

The following case and figure represent a typical case of measles:

A girl, six years old, after an incubation of ten days, was attacked with lachrymation, coryza, cough, and a temperature of 39.4° C. (103° F.). On the second day from the beginning of the invasion the temperature fell to 37.7° C. (100° F.). On the third day it rose again, and on the fourth day the temperature rose to 40° C. (104° F.), and an efflorescence, papular in character, appeared on the face and extended to the neck and thorax. The disease as represented in the picture is at the height of the stage of efflorescence on the fifth day from the beginning of the prodromal symptoms and the fifteenth from the date of the infection. It shows the swollen condition of the eyes, nose, and entire face. The photophobia was extreme, and there was considerable lachrymation, a continual, short, dry cough, and extensive coryza. The papules coalesced on the face, and were of a darker color than the widely separated lesions on the chest.

FIG. 127.



Typical condition of the face in measles. 15 days from infection. Female, 6 years old.

Plate VIII., facing page 564, represents a boy, eight years old, who was at the height of the efflorescence of an attack of measles.

The different stages of the typical lesions of measles are represented on his face and chest. The conjunctivæ are reddened, and the eyes, nose, and lips are swollen. The efflorescence in this case ran a very rapid course, beginning on the face in so intense a form that the desquamation had already appeared, although the efflorescence on the chest was in a much earlier stage of development. The papules and macules had coalesced on the cheeks and chin, while they still appeared as large, deeply reddened lesions on the forehead. On the chin and neck were areas of normal skin appearing like white blotches, their boundaries determined by the clusters of papules. On the side of the nose there was a slight desquamation, which had the furfuraceous character already described as typical of measles. On the chest the papules and macules were much smaller in size, of a much lighter color, and in some places had assumed a crescentic shape.

VARIATIONS IN TYPE OF MEASLES.—Measles during epidemics and in sporadic cases varies much in its type, and presents great differences in its prodromal stage, in its dermal lesions, in its desquamation, and in its entire course. Through a lack of appreciation of this fact the diagnosis of other diseases, such as rubella and various forms of erythema, is continually being made when, in fact, the disease represents one of the more unusual forms of measles. If these variations in measles were better understood, we should not find the disease rubella so often diagnosticated.

At times the stage of incubation of measles varies considerably. It may even be extended from the usual ten days to twenty-one days.

Instead of the usual prodromal stage, certain cases during epidemics of undoubted measles show few, if any, prodromal symptoms.

In addition to the usual catarrhal symptoms, in some cases there are vomiting and sore throat. Again, instead of a considerable elevation, the temperature may be scarcely above the normal degree. In addition to the other variations in the course of the prodromal stage of measles, cases have been noticed during epidemics in which the catarrhal symptoms were absent. Epistaxis of a mild form, and not apparently connected with the more severe types of hemorrhage, is sometimes met with. I have seen it only occasionally.

The efflorescence, which in the typical cases usually consists of papules, or vesicles and papules, may vary so as to simulate closely a common erythema, constituting the form called *lævis*, or may closely simulate a papular erythema. Again, the efflorescence may in certain cases be represented by minute vesicles or milia, characterizing the form called *miliarius*. Any of these forms may be confluent, but, as a rule, only upon the face.

There is another form of efflorescence which occurs in measles, which is rare, and of a more serious nature than the common benign forms which are met with ordinarily. This is called the *hemorrhagic* or *malignant* form, and is represented on the skin by small capillary hemorrhages. It is often rapidly fatal, and at times appears to be part of a general hemorrhagic diathesis represented by epistaxis, hæmaturia, and hemorrhages from other localities. The temperature in these cases is not typical, as it does not remit in the prodromal stage, thus depriving us of an important means of diagnosis; but a doubt as to the nature of the disease does not last long, as the other symptoms soon become prominent. The more prolonged the course of this form the better the prognosis, for if fatal it is usually quickly so. It may be complicated by a malignant bronchopneumonia.

The efflorescence, besides differing in its form, may vary to a great degree in its intensity. Thus, we may have every grade of papule or macule, from the smallest to the largest, and varying from a dark purplish to a light pink color. In like manner, although the arrangement of the efflorescence, especially on the chest, is somewhat crescentic, yet during epidemics of undoubted measles this crescentic shape is often absent. Instead of the efflorescence first appearing on the face and then extending to the thorax and extremities, we may find in undoubted measles that it begins first on the chest or some other part of the body; or the efflorescence may appear on the face and thorax simultaneously. We may also find that in certain cases the efflorescence appears first on the abdomen, or on the thighs, and yet the presence of other typical and undoubted cases of measles in the vicinity or in the same house assures us that we are dealing with the same disease. The efflorescence instead of lasting for a number of days may be evanescent and may subside within twenty-four hours. The entire absence of efflorescence is said to occur in some cases, but must be considered very rare, and its possibility has been doubted.

The desquamation of measles is of so light a grade that it is not surprising that in some cases no desquamation whatever is detected. Cases in which desquamation occurs without efflorescence are highly improbable, although such have been reported.

During certain epidemics of undoubted measles cases have not infrequently been noted in which the post-aural and cervical glands were enlarged.

There is a form of measles, called the *recurrent*, which is closely allied to relapsing fever. The main characteristic of this form is the high fever. The temperature will sometimes be raised for five or six days, will then become normal for seven or eight days, and will then rise again with a recurrence of the symptoms. This is a very unusual form, and one which needs merely to be mentioned here. It is accompanied by the general symptoms connected with the nose, eye, and bronchi which are met with in the typical form of measles.

Relapses have been reported to occur in measles, but they must be very uncommon. I have never met with such cases.

In reviewing the pictures which have been given of these variations, it must be evident that, although in the large proportion of cases measles runs so typical a course that the diagnosis is very easily made, yet such great variations in type are always so liable to occur that we should be extremely careful not to make a diagnosis of certain other diseases, such as rubella, except under unusual circumstances. This is important, because we know that during epidemics of well-marked measles all these great variations as to incubation, prodrome, efflorescence, desquamation, and the entire course not infrequently arise.

A case which occurred in my wards at the Boston City Hospital during an epidemic of measles which took place in that institution illustrates how greatly the symptoms and appearance of the disease may vary. The cases occurring in the hospital were almost without exception of the typical form, in which no mistake could be made as to the diagnosis of measles.

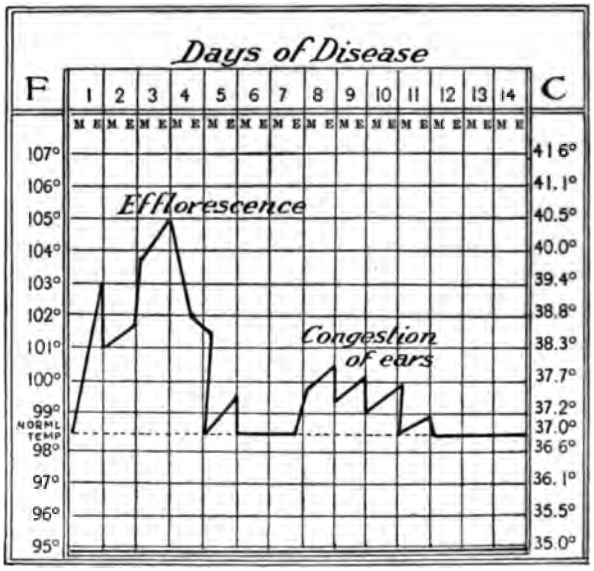
A girl who was in the hospital, and who was exposed to infection from the patients with measles, after feeling perfectly well on the previous day, was found in the morning to have slight coryza, cough, and a papular efflorescence not confluent even on the face, small in size, light pink in color, and not crescentic. While the efflorescence lasted the appetite was somewhat lessened, and the temperature was about 37.5° C. (99.5° F.). At the end of twenty-four hours the efflorescence had almost faded, and in a few days the general symptoms passed away, the patient's appetite had returned, the temperature had become normal, and she seemed perfectly well.

If this case had been met with as a sporadic one, it would have been impossible to make the diagnosis of measles, and from its mild nature it would have been supposed to be some slight form of disease, such as rubella.

I have met with cases of this type quite frequently, both during epidemics and sporadically; their cause is always obscure, and in them the diagnosis between measles, rubella, and papular erythema is often impossible.

The following chart represents the temperature during the stage of invasion and efflorescence in a typical case of measles, and also the accompanying mild congestion of the membranæ tympanorum which is so common in measles, and which in this case appeared on the eighth day and closely followed the efflorescence on the face:

CHART 24.



Measles with congestion of membranæ tympanorum during stage of efflorescence.

Complications and Sequelæ.—There are quite a number of complications and sequelæ which may occur in the course of measles. The most common of the serious ones are *pertussis*, *pneumonia*, and *tuberculosis*.

Pertussis.—Pertussis seems to have an intimate connection with measles, and its occurrence in the course of measles renders the prognosis more grave.

Broncho-Pneumonia.—The bronchitis which is so common an accompaniment of measles sometimes appears in a more severe form, attacking the smaller bronchi as well as those of medium size, and may result in a broncho-pneumonia, which is much more common as a complication of measles than is lobar pneumonia. The broncho-pneumonia does not, however, appear to be more severe when it arises as a complication of measles than when it occurs separately from that disease. Broncho-pneumonia as a complication of measles may occur very early in the course of the disease, even during the stage of invasion; but it occurs most commonly towards the end of the second week.

When, therefore, after the efflorescence has faded and the fever has subsided, the temperature again rises without evidence of local irritation in the throat, ear, or glands, we should suspect that a broncho-pneumonia is developing. The additional symptoms of quickened respiration and the movement of the alæ nasi will render still more probable the supposition that this complication is arising, even though nothing abnormal is detected in the lung itself. The absence of abnormal physical signs in the lung in the early stage of broncho-pneumonia is quite common. In infants the temperature of tubercular broncho-pneumonia does not seem to differ very much from that of ordinary non-tubercular broncho-pneumonia. The congestion of the larger bronchi, which appears to be almost a part of measles, may become subacute and chronic, instead of, as is usually the case, passing off soon after the maximum of the temperature and efflorescence.

Pleuritis may occur in the course of measles, but is not so common as pneumonia.

Otitis Media.—When an otitis occurs as a complication of measles it is characterized by the conditions described on page 582. In treating this complication the nose and naso-pharynx should be kept as clear as possible. The ear should be, as in the cases described in speaking of the treatment of the ear in scarlet fever, gently inflated by means of the Politzer bag, and the atropine solution (Prescription 80, page 570), together with dry warmth, should be used.

The following case illustrates this aural complication occurring in measles:

A girl, one year and seven months old, previously well, was attacked on March 6 with coryza, cough, lachrymation, a heightened temperature, quickened respirations, and a rapid pulse. An efflorescence of measles appeared on the face on the following day, and the child felt sick, coughed continuously, and had a hoarse voice. The respirations varied from 36 to 40, the pulse from 170 to 180. The skin was hot and dry, and the throat was somewhat reddened. In the afternoon the temperature in the axilla was found to have risen to 40.2° C. (104.8° F.). She vomited and had a convulsion. The nervous symptoms passed off in a few hours, free perspiration followed, and the cough became somewhat looser. At 8 o'clock in the evening the temperature was 40.1° C. (104.4° F.), the respirations were quickened, and the pulse was rapid. Nothing abnormal was found on physical examination of the chest. During the night she was somewhat delirious, and very wakeful and fretful. The temperature remained at about 40° C. (104° F.), the respirations were rapid, and the alæ nasi moved so perceptibly that it seemed as though a pneumonia must be developing. Frequent and careful examinations of the lungs, however, failed to show anything abnormal. She continued to be very restless during the night and the efflorescence appeared thickly on the abdomen and legs, but very slightly on the chest. She complained of pain in the chest from the continuous cough, but did not show any symptoms of pain or discomfort elsewhere. Towards morning it was found that an otitis of the left ear had developed, which in a few hours caused perforation of the membrana tympani. As soon as there was a free flow of pus from the ear the temperature fell to 38.8° C. (101° F.), the respirations became quiet and normal, the alæ nasi ceased to move, and the child fell into a quiet sleep. On the next day the efflorescence was pronounced all over the body, face, and extremities. From this time the measles ran its usual course, and was followed by desquamation and complete recovery.

The aural complication, however, proved to be very intractable, and although it was carefully treated, lasted for many months. The perforation of the membrana tympani did not completely heal for over a year, but the case finally resulted in complete recovery without any disturbance of hearing.

Eye.—In addition to the conjunctivitis which is a common accompaniment of measles, and which, as a rule, requires no treatment beyond the protection of the eyes from light, the inflammatory process may extend to the deeper tissues of the eye and cause other grave lesions, such as *blenorrhagic conjunctivitis*, *keratitis*, and *iritis*. These complications should be treated at once by a skilled oculist.

Tobeitz has called attention to the deleterious influence of measles in rendering more active any subacute or chronic affections of the eye which may have existed previous to the disease.

Thyroid Gland.—In a number of cases an acute swelling of the thyroid gland may take place during the course of measles. This swelling of the thyroid gland may even cause marked dyspnœa by pressure, but it usually disappears in two or three days. In some cases, however, a formation of pus has taken place, followed by destruction of a part of the gland. In intractable cases of this kind it has been found that the external application of iodine is useful.

Lymph-Nodes.—Enlarged cervical lymph-nodes are not so common in measles as in scarlet fever, but they may occur, and may even prove serious from the occurrence of suppuration.

Kidneys.—At times, at the height of the efflorescence, albumin may appear in the urine; but this is frequently merely a transient congestion of the kidney, due to the high temperature, and corresponding to the same condition in the period of efflorescence in scarlet fever. Nephritis may complicate measles, as it does scarlet fever, but it is comparatively rare.

Tuberculosis.—The most common sequela of measles is tuberculosis. This may occur either as a general miliary tuberculosis or as a tubercular disease of any of the organs or the joints. Tubercular disease of the joints seems to show a special predisposition to follow attacks of measles. It is noticeable that when a patient with a tubercular joint has an attack of measles the process in the joint is apt to become temporarily more active, and the prognosis is consequently more grave. The organ which in measles is most commonly affected by tuberculosis is the lung, and the most common form of tuberculosis of the lung is a tubercular bronchopneumonia. Tuberculosis of the lung may often occur as a sequela of measles when pneumonia has not been present. In infants the temperature of tuberculosis does not seem to differ very much from that of an ordinary broncho-pneumonia. In regard to the relation which exists between measles and tuberculosis, we should appreciate the danger, which seems to be a serious one, that the micro-organism of measles will render active an old and quiescent tubercular nidus, whether it be in the bronchial or the cervical glands or elsewhere.

The following case illustrates the infection of a patient with measles by the tubercle bacillus.

A girl, six years old, was always well until she had an attack of measles. Although there was no acute pulmonary affection following the attack of measles, she began to be affected with slight dyspnœa and a cough about one month after the measles had ended. A year later these symptoms increased, and she had swelling of the feet and complained of general malaise. She also lost considerably in weight and strength. On physical examination dulness was found at the apices of both lungs, and over the dull areas coarse and fine moist râles. Nothing abnormal was found in connection with the heart or kidneys. The temperature varied from 37.7° to 38.8° C. (100° to 102° F.), the respirations from 30 to 50, and the pulse from 120 to 130. An examination of the sputum showed the tubercle bacillus to be present.

Paralysis.—Another sequela, although a rare one, is paralysis. Cases thus complicated have shown mostly a paraplegia, and, according to Osler, frequently can be classified as post-febrile polyneuritis, although it is possible that some of them may be due to a rapidly ascending myelitis.

Noma.—A very rare sequela of measles is the disease noma (cancrum oris).

Among the rarer complications of measles are *empyema*, *endocarditis*, *pericarditis*, and *membranous laryngitis*. *Catarrhal laryngitis* and *tracheitis* are not infrequent accompaniments of the acute stage of measles. *Œdema* of the glottis is rare, but has been known to occur.

The irritation of the intestine, occurring commonly during the height of the efflorescence and temperature, sometimes becomes much more severe from the development of *colitis* as a complication.

RUBELLA.

Rotheln; German Measles.—It is now almost universally believed that there is, in addition to variola, varicella, scarlet fever, and measles, a highly infectious acute disease accompanied by an efflorescence on the skin which is distinct from these other members of the group of exanthemata. While we must recognize the propriety of mentioning the existence of this disease when speaking of this class of affections, we must also acknowledge that it is the weight of opinion, and not of proof, which has characterized rubella as a disease *sui generis*. The cause, the symptomatology, and the resulting diagnosis of rubella must be left for future investigation, until the special micro-organism which produces it and that which produces measles can be separated bacteriologically. The difficulty which arises in differentiating rubella from the other diseases of this class is chiefly in distinguishing it from measles. We cannot describe a typical case of rubella in such a way as to enable us to diagnosticate the disease in a sporadic case. On the other hand, this can be done so readily with the other exanthemata that we can at once diagnosticate a sporadic case.

Symptoms.—Rubella is described in many ways by observers in different localities, but is usually spoken of as essentially a highly infectious disease, with an incubation of two or three weeks, with slight or no prodromata, and with a slightly raised temperature, accompanied by mild catarrhal symptoms, and often by sore throat and swelling of the cervical and post-auricular glands.

Efflorescence.—The efflorescence is commonly described as papular or macular in form, of light grade, often evanescent, and seldom showing any desquamation. Forchheimer has described what he designates as an exanthem appearing on the mucous membrane of the fauces at the same time that the efflorescence appears on the skin. This exanthem has the same characteristics as that on the skin, excepting so far as it is modified by the difference in locality. Forchheimer believes that this exanthem is characteristic of German measles, and states that it is very short-lived, fades away within the first twenty-four hours, and is localized upon the velum of the palate and on the uvula, but rarely invades the hard palate or any other part of the mouth. The efflorescence consists of macular, distinctly pink-red spots resembling the roseola of typhoid fever, arranged irregularly, not crescentically, about the size of large pin-heads, very little elevated above the level of the mucous membrane, and with very little infiltration. These spots come out in their largest circumference, do not increase in size, and during their involution sometimes leave yellowish-brown spots or streaks. Koplik's spots, considered by him to be characteristic of measles, are said never to be present in German measles, but have been found by Widowitch ten times in a series of one hundred and thirty-five cases of rubella.

Complications or *sequelæ* following rubella are said to be uncommon. If a careful study is made of the variations which occur commonly during epidemics of undoubted measles, it will be seen that this description of rubella is one which may be applied to many mild cases of measles. As, however, epidemics arise in which these characteristically mild symptoms occur in many cases, and these cases may give rise to similar cases, it is probable that in the future a micro-organism distinctive of rubella will be found.

Diagnosis.—Bearing in mind the facts which have been mentioned, we can merely say regarding rubella that its diagnosis can seldom be made in a sporadic case.

Prognosis.—The prognosis is good.

Treatment.—The treatment is that of a mild case of measles.

VARIOLA.

Etiology.—Variola (small-pox) is one of the most virulent of the infectious diseases with which we have to deal, and is particularly fatal among infants and young children. The micro-organism which causes this disease has not yet been determined. It is characterized by severe constitutional symptoms, accompanied by a progressive efflorescence from macules and papules to vesicles and pustules, followed by the formation of crusts, these lesions having a tendency to result in cicatrices. Variola, in contradistinction to varicella, scarlet fever, and measles, is an extremely rare disease among infants and young children who have been vaccinated.

Although there are no characteristics of variola which are distinctive in children from those of the disease occurring in adults, it is important to recognize its chief features for the purpose of differential diagnosis. It is possible for the fœtus to contract the disease in utero. This, however, is rare, and it is well known that infants whose mothers are affected with variola can, even when born in small-pox hospitals, be protected from the disease if vaccinated immediately. The contagium is supposed to exist in the secretions and excretions, and to emanate from the exhalations of the lungs and from the skin. It is in all probability transmitted principally by means of particles of the crusts. It has a wonderful tenacity for clothing or any like means of conveyance. It has been proved that the contagium is active before the efflorescence occurs, though not so much so as later. It has also been fairly well proved that its activity ceases when all the crusts have fallen off and when the entire skin has become smooth. The most virulent form of the disease can be contracted from a mild form, such as varioloid.

Pathology.—The pathological conditions found in variola are chiefly those of the skin and the mucous membranes. The lesion begins as a round, somewhat raised macule, which develops into a hard papule, and later a small vesicle arises on its summit. This vesicle enlarges very rapidly and changes to a tensely filled pustule with a central depression. The size of this pustule corresponds to that of the original macule. Microscopically the macule consists of a circumscribed spot of hyperæmia in the capillary layer of the skin. The papule is formed, according to Weigert, by a sharply defined necrobiotic degeneration of the under layers of the rete mucosum, by which process the nuclei of the epithelial cells are destroyed. By the transudation of fluid into these areas the cells are pushed apart and the epithelial layer is lifted up as a whole, covering the area affected, and forms a vesicle, the inner part of which is composed of a mesh-work filled with lymph. In the vicinity of the necrobiotic focus an inflammation is set up, causing an increased growth of the cells of the rete which surround and wall in the focus on all sides. The developed pustule extends through the whole thickness of the cutis to the subcutaneous tissue. A net-work inside the pustule, which is most tense in the central part, connects the roof and floor of the pustule, and, in conjunction with the above-mentioned growth of the cells of the rete around the focus, causes the central depression. If the vesicle is pricked, only a part of the lymph flows out of the mesh-work within. The lymph is clear, and contains some white and red blood-corpuscles, streptococci and staphylocci, fibrin-flocculi, and molecular granules. The contents of the pustule are purulent, and those in the hemorrhagic form contain blood. Clumps of bacteria with analogous localized degeneration and its associated changes are found in the neighborhood of the pustules, also in the parenchyma of the internal organs and lymph-glands, as well as in the skin. When the variola has reached its height the central depression in the pustule disappears, because the increased tension in the contents tears away the mesh-work. The vesiculation begins in the upper central part and spreads downward towards the periphery. The pustule then collapses and changes to a crust, which after a certain number of days falls off, leaving a more or less deep scar covered with young epithelium. If the suppuration extends into this layer, scarring invariably results; it does not necessarily follow if the suppuration is confined to the upper layer. A distinct difference in the anatomy of a pustule of variola vera and one of varioloid does not exist.

On the mucous membranes of the mouth, nose, conjunctivæ, bronchi, œsophagus, rectum, sometimes the vagina, and also on the tonsils and the tongue, the same pustular efflorescence may be found, and is either superficial or extends more deeply. At times also a pseudo-membrane is found on the ulcers.

In the intestines swelling of Peyer's follicles is not uncommon. In the larynx the efflorescence may be associated with a fibrin exudate, and sometimes with œdema sufficient to cause death. Occasionally the inflammation extends deeper and involves the cartilages. In the trachea and bronchi there may be ulcerative erosions, but the characteristic lesions seen on the skin do not occur. There are no special lesions of the lungs, but congestion or broncho-pneumonia is very common.

Conjunctivitis, keratitis, and inflammation of other parts of the eye may occur in the course of the disease or afterwards.

Acute otitis media, with or without suppuration, is of common occurrence.

The pathological changes in the other organs consist of enlargement of the spleen and fatty degeneration of the liver, kidneys, and heart. Metastatic processes in the various organs and in the joints sometimes occur. In the hemorrhagic form hemorrhages in the various cavities, in the different organs, and, according to Golgi, in the medullary cavities of the bones, may occur, also in the serous and mucous surfaces and in the muscles. The blood shows an active leucocytosis during the pustulous stage, and the red blood-corpuscles tend to form in clumps instead of rouleaux.

INCUBATION.—The incubation of the disease varies from twelve to fourteen days, the latter being the most frequent period.

SYMPTOMS.—According as the symptoms of variola are mild or severe the disease has been divided into a number of forms, designated as follows: (1) *discrete*, (2) *confluent*, (3) *hemorrhagic*, and (4) *modified*. In all these forms the initial fever, convulsions, and general symptoms may be severe, and do not necessarily indicate which type of the disease is about to follow.

(1) **Discrete Form.**—The mildest and most typical form of the disease is that which is called *discrete*.

Prodromata.—In this form, the invasion, although sometimes less severe than in the confluent and hemorrhagic forms, is in infants and young children almost always of a grave type. In infancy and early childhood the disease commonly begins with convulsions. There may be vomiting, great restlessness, rapid pulse, high temperature, and in a number of cases the children quickly succumb to the disease from the virulence of the toxæmia. If they survive this early stage of the disease they usually present the same sequence of symptoms as in cases occurring in later life, but may eventually die from the exhaustion which often rises from a prolonged suppurative fever. In the prodromal stage the pulse is much accelerated and the temperature may be as high as 40°, 40.5°, or even 41.1° C. (104°, 105°, or 106° F.). In this stage we at times, especially among children, meet with an evanescent erythematous efflorescence. According to Simon, this manifestation is distinct from that of scarlet fever. It has a peculiar distribution and generally a limited extent, usually affecting the lower abdominal areas, the inner surface of the thighs, the sides of the thorax, and the axillæ; sometimes, however, it involves the whole surface. This efflorescence is distinct from the typical lesions of variola which occur later.

Efflorescence.—On the third or fourth day of the prodromal symptoms an efflorescence appears on the skin, and at this time the frequency of the pulse lessens, the temperature usually falls considerably, and the more severe symptoms improve, so that the patient appears much more comfortable. The efflorescence is at first represented by small red macules or papules, which, as a rule, first appear on the forehead, or on the face and mucous membranes, and later on the trunk and limbs. The papules are rather scattered in their distribution, and have a feeling as of shot under the skin. The macules when present soon become papules. On the third day by means of a good light a small vesicle can be seen at the apex of the papule, and by the fifth or sixth day the vesicular stage is well established and the vesicle becomes distinctly umbilicated. This appearance on careful examination can also be seen in the lesions of the mucous membranes. At about the eighth day the vesicles become pustules, the tops soon flatten, and the umbilication disappears, leaving an areola of injection and the intervening skin in a swollen condition.

Temperature.—The temperature at this time rises, from the suppuration which is taking place in the pustules. This rise of temperature is called the secondary fever, or fever of suppuration. The temperature remains high for from twenty-four to forty-eight hours, and then gradually falls until by the twelfth or thirteenth day it usually becomes normal. The contents of the pustules dry up, and crusts are formed. On the palms and soles small, hard disks form, which may of themselves fall off in infants, but in children as old as ten years would remain for a long time unless removed with the point of a knife.

Blood.—On examining the blood in cases of variola Arnheim found the hæmoglobin diminished at the beginning of the disease. After the formation of pustules and during their exsiccation, he found an increase of the hæmoglobin with diminution of the erythrocytes. When complicating suppuration occurred both the erythrocytes and the hæmoglobin remained for a long time abnormally diminished.

Pick in forty-two cases found no leucocytosis except in the stage of suppuration or in some complication like pneumonia. This was the case even when the temperature was high, the disease severe, and the termination fatal.

Desquamation.—By the fourteenth or fifteenth day the stage of desquamation is established. In some cases extensive scars are left on the skin where the crusts have fallen off. This is most apt to occur in severe cases.

(2) **Confluent Form.**—In contradistinction to the mild or discrete form of variola is the more severe form, called *confluent*, on account of the tendency of the lesions to coalesce. In the confluent form of variola the efflorescence usually appears at the same time as in the discrete form. At about the fourth day the lesions become confluent, the skin becomes reddened and swollen, and the face may be much distorted by the severity of the lesions. In this form the initial temperature does not fall to the same degree as it does in the discrete form, and, according to Sydenham, diarrhœa is likely to occur, particularly in children. The pharynx and larynx are especially apt to be involved, and the cervical lymphatics to be enlarged. The crusts adhere longer in the stage of desquamation than they do in the same stage of the discrete form.

(3) **Hemorrhagic Form.**—The third or *hemorrhagic* is the most virulent form of variola, and may occur in children as it does in adults, although not so frequently in the former as in the latter. Its symptoms in children are so severe that in almost every case it very quickly proves fatal. It is characterized by punctiform hemorrhages in the skin, appearing from the first to the fourth day of the prodromal stage, ecchymoses in the conjunctivæ, and hemorrhages from the mucous membranes. According to Osler, hæmaturia is the most common form of hemorrhage, hæmatemesis the next.

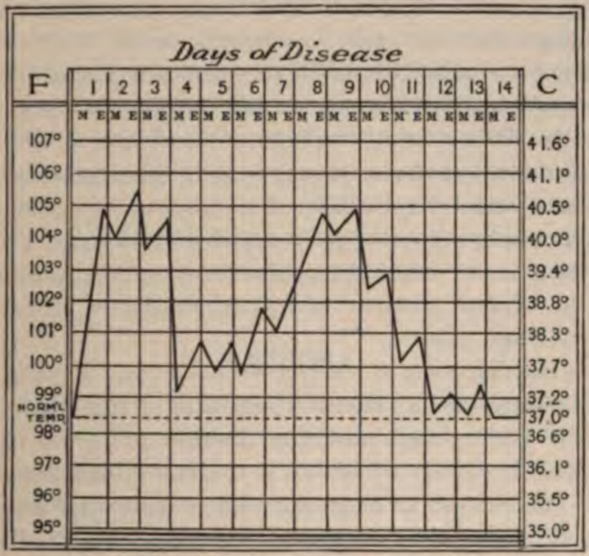
(4) **Modified Form.**—The fourth or *modified* form of variola occurs when the disease attacks individuals who have been successfully vacci-

nated. This form is called *varioloid*, but would be better termed *modified small-pox*. Modified small-pox is usually much milder in its symptoms than any of the other forms of variola, although the initial fever may be as high as in a severe case. The papules are fewer in number, the temperature becomes normal sooner, and the child seems comfortable in a shorter period of time, since there is usually no secondary fever from suppuration. The nearer the attack comes to the time when the child was vaccinated, the less severe will be the symptoms.

In any of these forms of variola the prodromal symptoms may be of a very severe nervous type, and this is especially characteristic of the disease as it occurs in children. For this reason variola may simulate other diseases in its prodromal stage, and may often cause death before the efflorescence has appeared. This is especially the case with the prodromal symptoms of the hemorrhagic form.

The following chart represents the usual temperature curve of the initial fever and the suppurative fever in a typical case of variola.

CHART 25.



Fever of invasion. Fever of suppuration. Variola.

COMPLICATIONS.—The most common complications of variola are those of the larynx and the lungs. When the larynx is affected, œdema of the glottis may suddenly arise and death take place from suffocation. When a lesion of the lung develops, it is usually in the form of a bronchopneumonia. Lobar pneumonia rarely complicates the disease.

In the throat the presence of the efflorescence occasions great irritation, and the accompanying secretions cause nausea and at times dyspnœa, with a cough which in weak children is very exhausting.

When acute inflammation of the middle ear has taken place the pain during the formation of the pus is very intense, but it subsides as soon as the sac bursts or is incised. This complication, therefore, requires early and careful treatment.

Although albumin is very frequently present in the course of the disease, nephritis is rare.

DIAGNOSIS.—There is no other constitutional disease accompanied by an efflorescence on the skin which in a typical case would be likely to be mistaken for variola. The severe constitutional symptoms, the slowly developing and rather scattered macules and papules, with the shotty feeling of the latter, the umbilicated vesicles gradually becoming pustules, the extensive crust formation, and the initial and suppurative fever, all render the diagnosis in most cases quite plain.

Extreme cases of varicella have been mistaken for variola. The differential diagnosis between the two diseases is given in the table on page 612.

Variola differs materially in its prodromal symptoms from measles, in which the pronounced catarrhal symptoms of the nose and eyes make the differentiation comparatively easy. Although the prodromal symptoms of scarlet fever and of variola, such as the convulsions and vomiting, are often of equal severity and somewhat similar, yet the pronounced symptoms connected with the throat in scarlet fever, and the appearance of an erythematous efflorescence, instead of the scattered papules of variola, serve to differentiate clearly the two diseases. We must, however, be careful not to mistake the evanescent efflorescence which occurs in the prodromal stage of variola for the erythema of scarlet fever. The distinction can usually be made by remembering that the efflorescence in variola has the peculiar distribution described under prodromata on page 597, while the typical location of the efflorescence of scarlet fever is first on the neck and chest.

In making the diagnosis of variola we must bear in mind the efflorescence which appears on the skin in the course of a general *vaccinia*. In vaccination the single lesion and the absence of severe constitutional symptoms make it hardly necessary to do more than refer to it in this connection. The differential diagnosis from general vaccinia is not difficult, and yet general vaccinia is so rarely met with that when it appears it almost always creates a suspicion that we may be dealing with variola. As a rule, in vaccinia the general symptoms are not severe, the disease being represented almost entirely by a slight malaise and loss of appetite, in conjunction with the appearance on the third or fourth day of an efflorescence on the skin, represented by papules, vesicles, and pustules, few in number and irregularly distributed, some on the face and nose and a few on the body and extremities. As these manifestations almost invariably appear after vaccination, this fact is of great aid in the differential diagnosis from variola. The subsequent course of a case of vaccinia is so much milder and shorter than that of variola that in a few days the differential diagnosis can be made easily.

PROGNOSIS.—The prognosis of variola depends upon whether the child has been vaccinated, and also upon whether children who have been vaccinated when infants have been revaccinated. In the unvaccinated the prognosis is always bad, and is proportionately grave the younger the child. In the vaccinated the prognosis is good, and is proportionately favorable to the shortness of the interval between the invasion of the variola and the time of the previous vaccination.

TREATMENT.—There is no specific treatment for variola, but it is of the utmost importance that the best hygienic care should be employed. The air of the room should be perfectly fresh. The crusts should be kept softened with a mixture of glycerin, oil, and carbolic acid, and the odor arising from them should be modified by the application of a dilute solution of carbolic acid.

In the initial stage of the disease stimulants should be freely given if the symptoms are severe, and the high temperature should be controlled by sponging with water at a temperature corresponding to the power of the child's reaction.

The greatest care should be taken during the stage of convalescence, and when the child is considered well the most rigid measures for preventing the spread of the contagium should be enforced. The clothing and everything connected with the child and its attendants, and the room in which they have been kept during the sickness of the child, should be thoroughly disinfected, the same precautions being taken to prevent the spread of variola as in scarlet fever. The immediate transferrence of a patient from its room to a small-pox hospital is in most communities considered the wisest method of dealing with the disease, and is usually enforced by law.

VACCINIA.

Vaccinia (cow-pox) is a specific eruptive disease occurring in cattle, especially in cows. The disease in human beings may be either a local affection, spreading from a point of inoculation, such as from vaccination, or may be a general manifestation arising from the entrance of poisonous matter into the blood. It is supposed that vaccinia may occur either by the introduction of the vaccine lymph into the blood or by auto-inoculation, and that the latter is probably the more common way. The disease is characterized by the appearance of papules, vesicles, and pustules of different sizes in different parts of the body and limbs, as well as on the face, and running a definite course. It may be said to be a rather rare disease. When its lesions follow vaccination they appear at about the fifth day after the inoculation. At the end of four days, however, minute vesicles can be seen with the magnifying-glass.

Vaccination.—By vaccination is commonly meant the inoculation with the virus of vaccinia as a preventive of variola. During a period of fifteen years no death has occurred from variola in Boston of a child who had been vaccinated before it was five years old. During the same period the percentage of deaths among the unvaccinated at the Boston Small-Pox Hospital was seventy-five per cent., while that of the vaccinated was three per cent. For the past seventeen years no person who has been successfully vaccinated within five years has died of variola, and those who have been attacked by variola have had the disease in a very mild form. In order to show the relative frequency of deaths occurring in the vaccinated and in the unvaccinated I have arranged the following table, based on a report by Dr. Barry of an epidemic of variola at Sheffield, England, during 1887 and 1888. The table shows the percentage of those who, living in houses invaded by variola, were attacked by the disease, and also how many of these died. It also gives the percentages for all ages, and for those under ten years and over ten years.

TABLE 66.

Individuals living in Houses invaded by Variola.

		1. All ages. Per Cent.	2. Over ten years. Per Cent.	3. Under ten years. Per Cent.
Vaccinated......	Attacked............	23.0	28.1	7.8
	Died................	1.1	1.4	0.1
Unvaccinated....	Attacked............	75.0	68.0	89.9
	Died................	37.2	37.1	38.1

The low percentage of children as shown in column 3 is very striking in comparison with column 2, which represents older individuals, and enunciates the importance of revaccination. It also impresses upon us the significance of the difference in the number of deaths between the vaccinated and the unvaccinated. When large numbers of cases of variola have been reported figures show that among the vaccinated nineteen out of twenty recover, while of the unvaccinated fifty individuals out of one hundred die. It is therefore evident that vaccination is highly protective against variola, and physicians should insist on the vaccination of every individual in the community. One vaccination, however, does not protect for a lifetime. On the contrary, revaccination is just as important as the primary operation. One attack of variola does not always protect an individual from a second invasion, and more should not be expected from vaccination.

Revaccination should be performed at intervals of eight or ten years, and in a shorter time when cases of variola appear in a community. The danger of serious results arising from vaccination is extremely small.

The time at which vaccination should be done is of considerable importance. The infant should be vaccinated early, before it begins to be exposed to the danger of contagion from sources outside of its home. We must, however, remember how low is its vitality at birth, and how readily this vitality is affected by what would be considered trifling conditions for the older child or for the adult. A time should be chosen when the infant is not subject to the other disturbing conditions which naturally arise in the first two years of life, such as weaning and the irritation of the dental periods. If it is found necessary to vaccinate the infant after the sixth or seventh month, or before the twentieth, it should be done in an interdental rather than in a dental period, and not at the time when its food is being changed, or when it is suffering from either slight catarrhal conditions or some definite disease. I prefer to vaccinate the infant when it is four or five months old,—that is, just before the period when the first tooth appears. At this age it has usually become accustomed to its food, its digestion is in equilibrium, and its vitality is much above what it was in the early weeks of its life. By the fifth month also it will usually have developed the outward symptoms of syphilis if it has inherited that disease from its parents.

The vaccine virus can be introduced into any part of the body through the skin, and according to the fancy of the physician or parents. Girl infants can be vaccinated just below the knee on the outer side of the leg, so as to avoid having a scar on the arm, to which women usually object. I am accustomed to vaccinate boys on the outer side of the upper arm. Whether the vaccination is performed upon the leg or the arm, we should first inquire if the person who is to take care of the infant is right-handed or left-handed. If the nurse, for instance, is right-handed, she will naturally hold the infant on her left arm, and in this case, the infant's right arm being towards the nurse, it is better for the vaccination to be on the left arm. The process should be reversed when the nurse is left-handed, and in this case, for the same reason, it is better to vaccinate on the right arm or leg. The form of virus which I have been accustomed to use, and which I consider the best, is taken from cows rather than from human beings. It should be very carefully prepared by those who have made a scientific study of the subject, and, if possible, on farms which are under State supervision.

It has been pretty well proved by careful observation of large numbers of primary vaccinations that those who in later life contract variola have the disease in a less severe form when in their primary vaccinations they have been inoculated in three places at once rather than in two, and in two places at once rather than in one. The general constitutional disturbance also does not appear to be greater when the inoculation has been in two or three places rather than in one. The evidence therefore seems to be in favor of inoculating in two or three places in primary vaccinations. A very small surface is amply sufficient for the proper introduction of the virus. A pointed ivory quill charged with glycerinated lymph (such as is shown in Plate IX.) can be used directly for removing the epithelium, for exposing the smaller blood-vessels, and for introducing the virus. I prefer not to use any more instruments than possible, in order to avoid the possible introduction of some foreign substance which might interfere with the natural course of the vaccine virus and cause unnecessary inflammation.

The utmost precaution should be taken to insure against infection from other micro-organisms besides the vaccine virus. The part of the skin chosen for the vaccination should be thoroughly washed with soap and water and with alcohol. The hands of the operator should be clean and aseptic. A series of short scratches should be made about one-half centimetre (about one-fourth inch) long, four or five in number, and in two sets, one crossing the other, until the epithelium is sufficiently removed to show that the blood-vessels are exposed, but not to a degree that bleeding should take place, for in the latter case the virus may be prevented from gaining an introduction to the general circulation. The point of the quill should then be dipped into water which has been freshly sterilized. The flat part of the quill which is charged with the virus is then thoroughly rubbed into the wound. The skin should be protected for four or five minutes from contact with anything and then sealed with aseptic cotton and collodion. After two days this cotton can be removed, and, unless the subsequent lesion is broken, this measure is an additional safeguard against infection from extraneous matter in the first few days.

Plate IX. shows the different stages of a vaccination as they occurred in one case carefully observed by the artist and myself every day.

On the fifth day, as shown in the plate, a round, clear vesicle was seen at one end of the vaccination scratch, while at the other end there happened to be left a little brown crust.

On the eighth day an irregular-shaped lesion about ½ cm. (¼ inch) long, and 1 cm. (½ inch) wide, somewhat depressed in the middle, and with a clear vesicular border, appeared.

On the tenth day the lesion had increased in length to 2 cm. (⅞ inch) long, and to a little over 1 cm. (½ inch) wide, and there was an erythematous condition of the skin forming an areola with a diameter of about 2 cm. (⅞ inch), in the middle of which was the lesion just described. This areola was of a light shade of red, and on its outer border were irregularly distributed little light red maculæ.

On the twelfth day very nearly the same appearances were present as on the tenth day, except that the areola was very much more intense in its red color, and had grown to the size of a circle 3 cm. (1¼ inches) in diameter. Some of the little maculæ had become vesicles.

On the sixteenth day, in place of the vesicular lesion with its depressed centre, a crust had formed with a narrow line of redness around it, and on the outer border of this areola the redness gradually became fainter and shaded off into the normal skin.

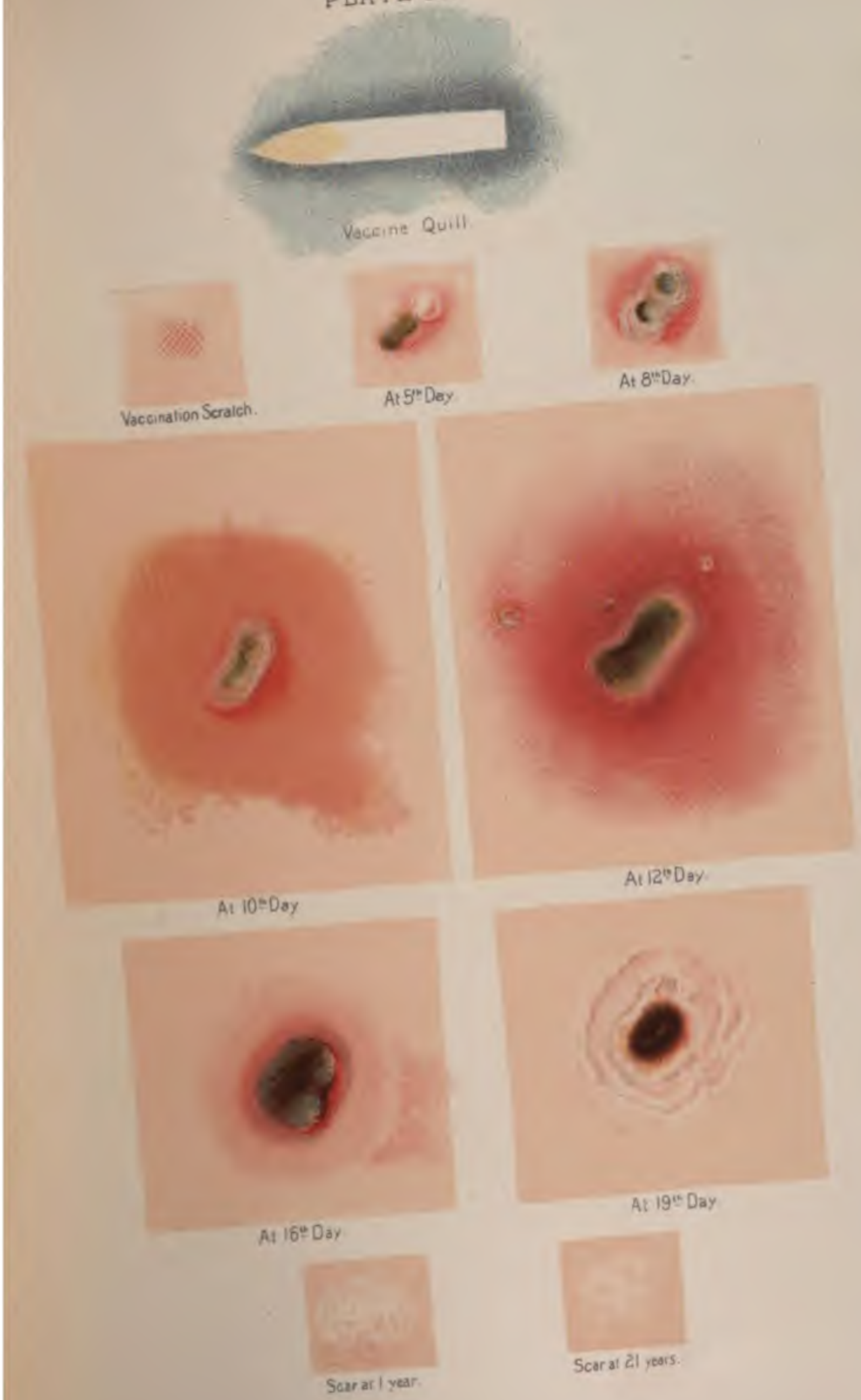
On the nineteenth day the crust was smaller, the redness had disappeared, and where the areola was most pronounced there was a slight desquamation.

A vaccination scar at *one year* and *twenty-one years* is also shown in the plate.

Every case of vaccination does not present exactly the same appearances. The lesions may differ in shape and size, and one individual may be affected more intensely by the virus than another; one may have accompanying severe constitutional symptoms and another have none. The chain of lymphatics may be affected as far as the axilla or the groin.

As a rule, the following description represents pretty well the usual course of the disease. After the vaccination, the skin shows nothing new

PLATE IX.



Vaccine Quill.

Vaccination Scratch.

At 5th Day.

At 8th Day.

At 10th Day.

At 12th Day.

At 16th Day.

At 19th Day.

Scar at 1 year.

Scar at 21 years.

until the third, fourth, or even fifth day, when a small red point appears. This soon becomes a papule; by the next day a vesicle is developed; about the sixth day this vesicle usually becomes umbilicated, and is surrounded by a faint red zone. By the eighth day the vesicle is fully developed, and by the ninth day the red zone increases rapidly and the vesicle soon becomes a pustule. By the eleventh or twelfth day a crust is formed, and this crust falls off from about the fourteenth to the twenty-first day, in some cases an ulcer being left which heals by another crust being formed, in others the skin remaining intact. From the eighth to the twelfth day there may be a slight amount of fever and coated tongue, with some loss of appetite, and the glands of the axilla or groin may become enlarged and tender. The scar, although perhaps not typical, can usually be recognized by its small depressions (pits) and its location.

In some cases, instead of the healing of the scratch in a few days, or the formation of the vesicle of a successful vaccination, irregular excrescences of a fungus-like character may appear. These in all probability have no connection with the true vaccine virus, and are not protective. In addition to the rather rare cases of vaccinia, various efflorescences at times appear on the skin, not only in the neighborhood of the vaccination lesion, but also in other parts of the body. They may be present on the fourth or fifth day, or even later, in the second week, and are probably caused by some reflex connection with the vaccination lesion. They vary considerably in form, but are usually represented by a multiple or papular erythema or an urticaria. It should be remembered, when an unvaccinated child has been exposed to variola, that if it is vaccinated within forty-eight hours it will probably be protected, and if within five or six days the variola poison will be so modified as to produce only a mild form of the disease.

VARICELLA.

Varicella (chicken-pox) is the mildest in its symptoms and the most favorable in its prognosis of the whole group of exanthemata. It is highly infectious, and is characterized, in distinction from the other exanthemata, by its long stage of incubation, the shortness or absence of any prodromal stage, its vesicular efflorescence, and the absence of sequelæ.

ETIOLOGY.—Varicella has been known as an independent disease for the last two centuries. At one time it was not clearly differentiated from measles and scarlet fever, and in some parts of the world it is supposed to be closely allied to variola. This opinion, however, is not generally substantiated, and we can accept varicella as a distinct disease. It can occur at any age, but the most common time for its appearance is in the middle and latter part of the first year. It continues to be a common disease all through the early and middle years of childhood. The susceptibility to the contagium of varicella lessens after ten years of age, and almost disappears at puberty. It is sometimes sporadic and sometimes epidemic. It occurs with equal frequency at all periods of the year. The vehicle of contagium is not known, but it probably enters the system by the lungs. The specific organism which produces varicella has not yet been determined.

PATHOLOGY.—Deaths from varicella are so extremely rare that our knowledge of the pathology of the disease is necessarily limited. It is evident, however, that the efflorescence of vesicles, which represents the principal morbid lesion of the disease, is of a somewhat different type from that which occurs in variola. The vesicle is much nearer the surface than in the latter disease, being formed mostly by the upper layers of the epithelium. The vesicle itself is seldom multilocular, a condition which is frequently present in variola. The contents of the vesicles are usually a clear serum, the progression to a pustule being rare in comparison with the lesion of variola. The lesion so rarely involves the deeper layers of the skin, and the process is usually so very mild, that it is seldom that sufficient destruction of the tissue takes place to produce a scar.

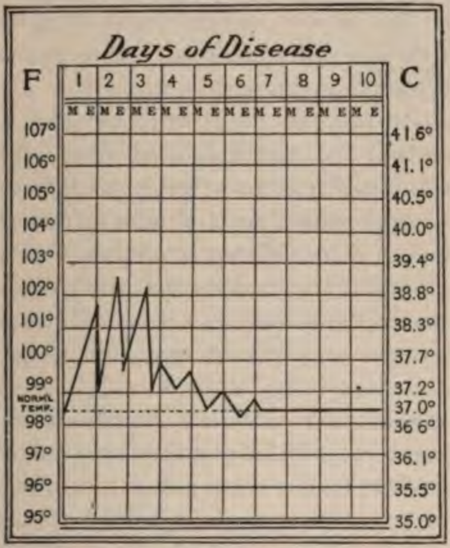
The lesions may appear on the mucous membranes as well as on the skin. At times the lesions assume a much more serious form and may become gangrenous. In *gangrenous varicella*, according to Eustace Smith, the vesicles, instead of drying up in the ordinary way, become black and larger, so that a number of rounded black crusts are scattered over the surface of the body. If a crust be removed, it is found to cover an ulcer more or less deep. Around it the skin is of a dusky red color. All the vesicles do not become gangrenous, so that we find crusts of the ordinary appearance mixing with the blackened crusts. The gangrenous process often penetrates deeply through the skin to the muscles. The lesions at times are so extensive as to form ulcers which may invade and destroy large areas of tissue.

INCUBATION.—The stage of incubation is variable, but lasts from eight or ten days to three weeks, the usual time being about seventeen or eighteen days.

SYMPTOMS.—*Prodromata.*—There are rarely any prodromata in varicella, beyond a slight malaise for a few hours. At times, however, especially in young infants, the onset of the disease may be severe: it may be characterized by vomiting, and, when the temperature is high, even by convulsions. In rare cases the prodromal stage is of considerable length and the prodromata resemble somewhat those of the other exanthemata.

Efflorescence.—The disease usually shows itself in the form of an efflorescence, the characteristic and most common lesion of which is a vesicle. The lesion, however, is in the beginning a macule, which quickly becomes a papule, and the papule so rapidly develops into a vesicle that it is in the vesicular stage that we usually first notice the efflorescence. These macules and papules are so superficial that they are soft to the touch and do not give the shotty feeling which is so common in these lesions when they occur in variola. The vesicle of varicella, as a rule, is not umbilicated, and its contents but rarely become pustular. It may be surrounded by a light red areola, but this is not present in all the lesions. The usual course of progression in the lesions is that the vesicle flattens, its contents are dispersed on the skin or absorbed, and a small crust is formed, which finally falls off, leaving the skin smooth and without a scar. Occasionally a scar results from some individual lesion in which the inflammatory process has involved the deeper layers of the skin. The efflorescence is irregular and general in its distribution, the lesions appearing on the face and head, in my experience especially behind the ears, on the body, usually first on the back, and finally on the extremities. It comes out in successive crops, so that very different lesions may be found on the skin at once, representing the early and late manifestations of the efflorescence. It, however, may first appear in the throat, but is not so often seen in this location as is the efflorescence of scarlet fever or measles. It is possible that the efflorescence always appears first in the throat, but that in many cases it is not seen early enough to be recognized, as the manifestations are very evanescent.

CHART 26.



Varicella simplex.

This efflorescence of varicella is almost the only one which is characteristic of a specific disease. By this I mean that while a vesicle does not necessarily allow us to diagnosticate any disease of the skin, yet when these vesicles with their areolæ, in combination with constitutional symptoms, appear in groups in different parts of the body, there is no other disease with which we should be likely to confound it, with the exception of variola, vaccinia, and possibly herpes zoster.

The temperature in varicella is in most cases not high, and is very irregular. It usually rises when a crop of lesions of any considerable number develops, and falls again at the outbreak.

Chart 26, on page 607, shows the temperature in a typical case of varicella simplex.

Diagnosis.—The diagnosis of varicella is not difficult if we bear in mind the characteristics of the diseases which it is most apt to simulate.

From Variola.—In differentiating it from variola we must consider the great difference in the rapidity of the development of the efflorescence in the two diseases. In variola it is essentially slow, in varicella it is characteristically quick. The papules of variola are hard to the touch, those of varicella are soft. The vesicle of variola, as a rule, is umbilicated and soon becomes a pustule ; these characteristics are absent in varicella. The whole course of variola occupies a period of from two to three weeks ; the course of varicella is much shorter, and is often limited to one week. Finally, the severe constitutional symptoms and the long prodromal stage in variola differ essentially from the lack of prodromata and the mild constitutional symptoms in varicella.

From Vaccinia.—In vaccinia the slow progression of the lesions from papules to pustules, and the rather limited areas affected, serve to distinguish it from the successive crops of vesicles, with their rapid development and extensive areas, which are met with in varicella.

From Herpes Zoster.—The differential diagnosis of varicella from herpes zoster is not difficult, if we consider that the vesicular efflorescence in herpes zoster follows the course of some set of nerves, while that of varicella is perfectly irregular and is in no way connected with the distribution of the nerves.

The course of varicella is rapid. It is characterized by a sudden onset of constitutional symptoms, with the almost immediate appearance of the efflorescence. The efflorescence runs a rapid course, appearing quickly on different parts of the skin, and disappearing almost as quickly as it appears. The disease lasts about a week or ten days, and, as a rule, has no serious sequelæ. It is rarely complicated by any other disease.

Complications.—During the course of certain epidemics, however, it has been noticed that the *kidney* is affected. This complication usually occurs after the efflorescence has almost disappeared, and in the second week from the time of the beginning of the attack. In these cases albuminuria is present, and in all probability is caused by some form of nephritis, although nothing definite is known about this class of cases.

Gangrenous Varicella.—A complication which at times arises in varicella is what is called the gangrenous form of varicella. Although it is most common in ill-nourished children, yet it does not necessarily attack this class of cases, and it seems to have some connection with the gangrenous processes which certain individuals show a tendency to develop.

The following table gives the chief points of difference between varicella and variola :

TABLE 67.

	Varicella.	Variola.
Incubation	Two to three weeks.	One to two weeks.
Prodromata	None or slight.	Three to four days in length. Active. Severe.
Efflorescence	Rapidly becomes vesicular. Not umbilicated. Unilocular. Irregular. Lesions numerous. Universally distributed in successive crops. Vesicles differ greatly in size. On pricking, collapses entirely.	A slow progressive development from a macule to a papule, from a papule to an umbilicated vesicle, then to a pustule. Multilocular. Regular. Lesions not numerous. Defined in its localization. Lesions, as a rule, of uniform size. On pricking, collapses partially.
Desquamation	Slight crust formation.	Pronounced crust formation.
Duration	Short, one week to ten days.	Long, three to four weeks.
Type	Mild.	Severe.
Temperature	Irregular, not high.	Rises suddenly. Remains high until papules are developed, when it falls considerably. Rises again during the development of the pustules.

Prognosis.—The prognosis of varicella is usually, unless the above-mentioned complications arise, extremely favorable. Cases occur in which the prognosis is rendered unfavorable by lack of proper care during the convalescence, resulting in broncho-pneumonia and other diseases. In some cases the prognosis is rendered unfavorable by the anæmia which is apt to follow an attack of varicella, and is at times pronounced.

Treatment.—The treatment of varicella is simply symptomatic. The child should stay in the house, and its room should be kept at an even temperature. The diet should be milk. The child should be carefully watched to prevent it from scratching, as lesions deep enough to produce scars may often be obviated in this way. This treatment should be continued until all the constitutional symptoms have passed away and the efflorescence has disappeared. Complete isolation should, if possible, be enforced, as, although the disease is usually insignificant, we can never in the beginning determine whether or not a rare and severe case is about to develop.

These rules for treatment are precautionary, and are based on the supposition that a child who has had a constitutional disease of this nature must be more sensitive to exposure of various kinds. As it is possible in some cases for the kidney to be affected in the later stages of the disease, just as it is in scarlet fever, it is well to guard against this complication by the protection of the skin from changes of temperature and by the use of milk as a diet. In a considerable number of cases, especially in young children, an anæmia of greater or less degree results from the disturbance of nutrition which so often accompanies the disease. In these cases the administration of saccharated carbonate of iron or of tartrate of iron and potash is indicated.

The following case represents an ordinary attack of varicella. A boy was brought to the hospital to be treated for a supposed cold. On examination nothing abnormal was found except the lesions shown in the throat in Plate X.

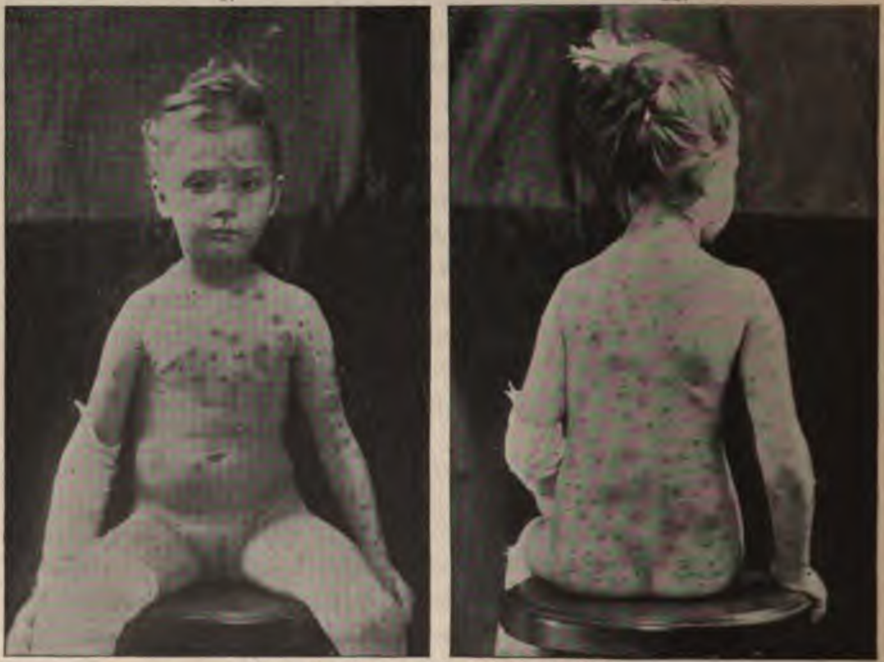
The tongue was very slightly coated. The tonsils were not enlarged. The mucous membrane of the hard and of the soft palate and of the pharynx was slightly hyperæmic. On the upper and right side of the hard palate and very near where it joined the soft palate were two small vesicles surrounded by distinct red areolæ. To the left and below these lesions were three minute macules, two of which had almost become papules.

This case illustrates very well the importance of making a thorough examination of the throat in children, for unless the throat had been examined this child would have been supposed to have had a cold and would have been allowed to remain in the clinic and thus spread the contagium.

The following case illustrates still further the efflorescence of varicella when at its height and on the second day. The efflorescence on the back is shown in Plate X.

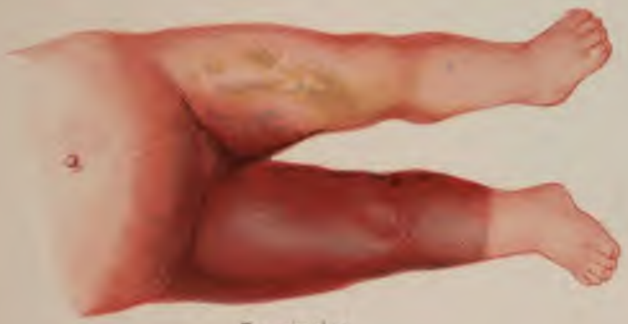
A girl was attacked with headache and malaise in the morning. In the afternoon an examination showed an efflorescence in the throat, but there was also a well-marked vesicular efflorescence on the back. This efflorescence soon began to come out in crops in different parts of the body, on the limbs, behind the ears, and on the scalp. There were also a few lesions on the face. On the back were a number of lesions, some of which were simply macules, and again a few of the macules had become papules. In most

Fig. 128.

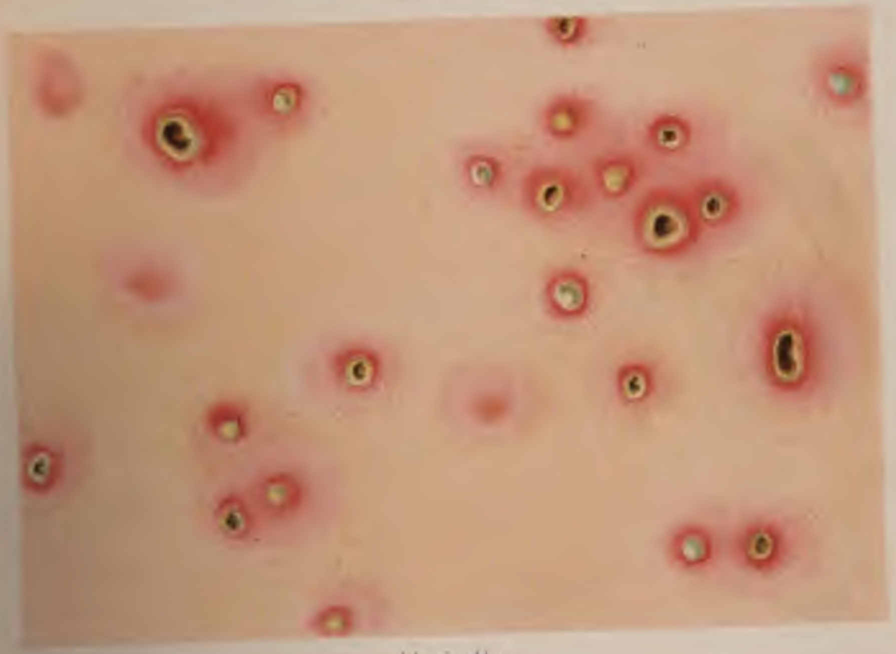

I. II.

Varicella. Stage of efflorescence, third day.

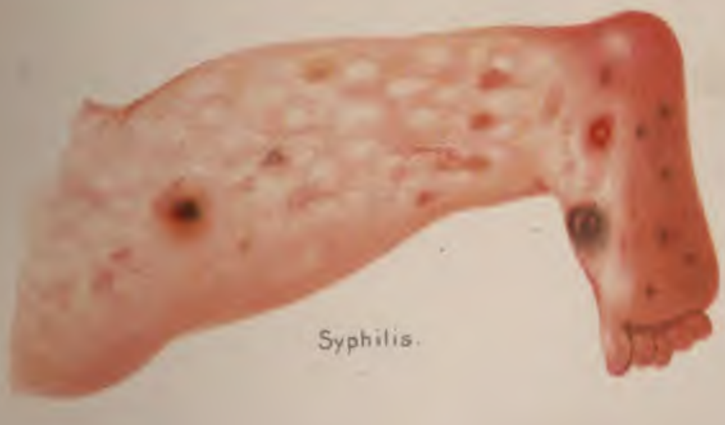
PLATE X.



Erysipelas.



Varicella.



Syphilis.

cases, however, the lesions were distinctly vesicular, varying in their contents to such a degree that sometimes they had a pearly white appearance and again the yellowish color of a vesicle which had become somewhat pustular. In other places the vesicles had broken down and little crusts had formed in their centres, which were somewhat indented. On pricking one of these vesicles it collapsed and was emptied of its entire contents, showing that it was unilocular. The vesicle of variola when pricked in this way would in most cases be only partially emptied, showing that it was multilocular.

The lesions of varicella are well represented in Fig. 128.

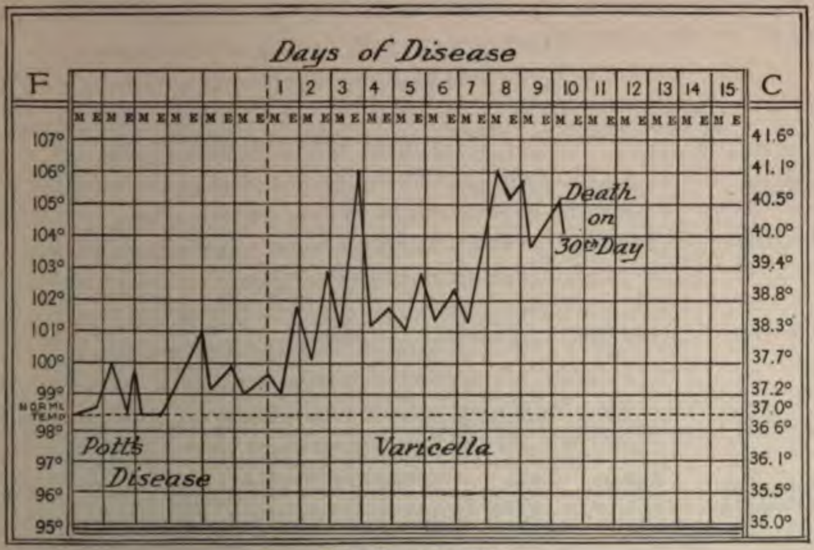
This child was brought from the surgical ward, where it was being treated with plaster-of-Paris bandages for an injury to the arm. The efflorescence, chiefly vesicular in character, first appeared behind the ears. The whole of the child's back was thickly covered with the efflorescence. The lesions were also on the arms, legs, abdomen, and front of the chest. They also appeared on the chin, lips, face, nose, forehead, and scalp.

The plaster bandage was removed, as a fixed bandage should never be used during the course of any of the eruptive diseases, owing to the probability that extensive ulcerations will develop under them.

The following case and chart are illustrative of gangrenous varicella:

A child, three years of age, was brought to the hospital with Pott's disease, and with a paraplegia arising from a transverse myelitis caused by the disease. Nothing abnormal was found in connection with the lungs, heart, or kidneys. Until the child was attacked with varicella the temperature was usually normal, but sometimes rose to 37.7° C. (100° F.), and occasionally as high as 38.3° C. (101° F.).

CHART 27.



Varicella gangrænosa.

About one month before entering the hospital the child became restless, and his temperature rose somewhat. On the following day the symptoms became more marked, and the temperature was found in the evening to be 39.4° C. (103° F.) On this day an efflorescence of varicella appeared on his skin. During the third day of his sickness his face swelled, and in the evening his temperature was found to be 41.1° C. (106° F.). The vesicular efflorescence was well developed on his trunk and face by this time. Somewhat later it became universal and assumed a purulent character, especially about the face. During the fourth, fifth, and sixth days of the disease his temperature varied in the evening from 39° C. (102.2° F.) to 39.4° C. (103° F.). On the seventh day of the disease all the symptoms increased in severity, and the temperature was found to be 41.1° C. (106° F.) On this day some of the vesicles on the face had become ulcers. Nothing abnormal was found in the lungs, and no albumin or casts in the urine. A psoas abscess developed during the progress of the varicella. The ulcers on the face extended to such a degree that the child lost the sight of one of its eyes. The child sank rapidly. The treatment, which was essentially with stimulants, failed to keep up its strength, and the local treatment in connection with the eye proved entirely unsuccessful.

The child continued to grow weaker, and died on the thirtieth day from the time when the first symptoms of the varicella were noticed. No autopsy was obtained.

DIFFERENTIAL DIAGNOSIS OF THE EXANTHEMATA.

The characteristics of this group of exanthemata are as follows. In none of these diseases has the specific organism been determined. When it shall have been, its detection will enable us to state definitely what disease we have to deal with, and even in the atypical cases of measles we shall be able to decide whether it is a case of true measles or some disease such as rubella, which closely simulates its irregular forms. The following table presents the chief points of differential diagnosis in the exanthemata:

TABLE 68.

	Scarlet Fever.	Measles.	Rubella.	Variola.	Varicella.
Incubation.....	4 days.	10 days.	21 days.	12 days.	17 days.
Prodromata....	2 days.	3 days.	A few hours.	3 days.	A few hours.
Efflorescence...	Erythema.	Papules.	Papules.	Macules. Papules. Vesicles. Pustules.	Vesicles.
Desquamation..	Lamellar.	Furfuraceous.	....	Large crusts.	Small crusts.
Complications and sequelæ..	Kidney. Ear. Heart.	Eye. Lung. Tuberculosis.	....	Larynx. Lungs.	....

In addition to the leading points which are indicated in the table, the general symptoms, the topography, and the temperature of these diseases distinguish them from each other.

The slow progressive development of variola is very distinct from the acute, rapid course of all the others. The vomiting and sore throat of scarlet fever are usually quite distinct from the coryza, lachrymation, and cough of measles. In variola the rise of temperature during the prodromal stage, its decided lessening at the time of the appearance of the efflorescence, and its gradual rise again during the stage of suppuration, are very distinct from the sudden rise of temperature in scarlet fever during the prodromal stage and up to the height of the efflorescence. In like manner the temperature in measles differs from that of the other diseases in its sudden rise on the first day of the prodromal stage, in its lessening on the second day, and in its rise on the third day and up to the height of the efflorescence. The manner of the decline of the temperature differs in variola, in scarlet fever, and in measles. While in variola it is slow and prolonged, in scarlet fever it is rather rapid, although it declines by lysis, and in measles the fall is often by crisis. In contradistinction to variola, scarlet fever, and measles, varicella and rubella differ markedly in the absence of a prodromal stage, in their short duration, and in their evanescent and moderate temperature.

PAROTITIS.

Etiology.—Parotitis (mumps) is a highly infectious disease which attacks the parotid gland. No specific organism has been determined. Its period of incubation is usually from two to three weeks, but cases in which the period was only three or four days have been reported.

Symptoms.—The onset of the attack is usually accompanied by a sense of chilliness, a rise of temperature, and a sensation of stiffness and tenderness about the jaws. This is succeeded by a swelling in the region of the parotid gland, which becomes enlarged and tender, rendering deglutition difficult and often very painful. The disease begins on one side, but the other gland is usually involved in a day or two. The infection is sometimes confined to the submaxillary glands on one or both sides.

The duration of an attack of parotitis is from a few days to a week, but the infection may last for two or three weeks, and it has been stated in some cases to antedate the appearance of the glandular enlargement. In boys at the age of puberty the complication of orchitis at times arises.

Although the symptoms of parotitis are commonly very mild, unusual cases sometimes occur in which the children are quite sick, and there have been instances in which the orchitis was of so high a grade that acute delirium supervened, and in one case reported by Dukes the boy fainted when the orchitis began.

Exceptionally, some cases of parotitis are marked by general malaise with little or no pain.

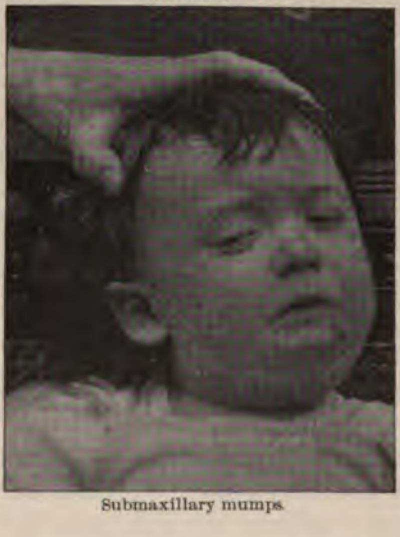
Diagnosis.—It is sometimes difficult to differentiate parotitis from a simple non-infectious enlargement of the parotid gland or of the glands in its neighborhood. When the parotid gland is enlarged it usually shows a characteristic swelling under and behind the lobe of the ear, so that the lobe is pushed somewhat upward and forward. This swelling increases rapidly, is very tender, is not especially reddened, does not fluctuate, and is accompanied by constitutional symptoms. The diagnosis is readily made if after a few days the unilateral swelling is followed by corresponding symptoms on the opposite side.

Treatment.—There is no especial treatment for the disease, as it is self-limited and runs a definite course. The children should be carefully isolated, in order that there may be no further spread of the affection. As deglutition is painful, their diet is usually milk and soups. They should be carefully protected from exposure, and should be confined to their rooms. Older children should be confined to bed, as orchitis in boys and trouble with the mammæ in adolescent girls are less likely to arise under these conditions. It is usually better to apply some soft cotton-wool to the painful swelling, and to protect it from any irritation.

Submaxillary Mumps.—The submaxillary glands are enlarged in children from various causes. At times the enlargement of the glands is accompanied by pain and tenderness, constituting a disease which has been called submaxillary mumps. In the beginning, however, we should not at once make this diagnosis, as the glands may become enlarged and tender from various causes which have no connection with the specific disease mumps.

Fig. 129 represents a probable case of submaxillary mumps.

Fig. 129.



Submaxillary mumps.

The child, a girl two years and four months old, was suddenly attacked with a swelling of the submaxillary glands, accompanied by pain and a slight amount of tenderness over the swollen region. She had a history of exposure to parotitis. On the following day the swelling had extended under the entire chin and up the left side of the neck to the face and ear.

The swelling, pain, and tenderness lasted for a number of days and then gradually subsided. Nothing more definite was discovered regarding the case.

The diagnosis in a case of this kind must be held in abeyance for a few days, and strict isolation should be enforced, as, if the cause of the glandular enlargement proves to be infectious, other children should be protected.

DIVISION IX.

DISEASES OF THE MOUTH, NOSE, EAR, NASO-PHARYNX, AND PHARYNX.

DISEASES OF THE MOUTH.

Nomenclature.—Much confusion exists as to the nomenclature of diseases of the mouth. A great variety of names has been used by different authors to describe the same disease, so that at times it is quite difficult for one investigator to compare his work with that of another. Such terms as "canker" and "aphthæ" are so commonly used for almost any morbid condition of the mucous membrane of the mouth that they have ceased to convey any definite idea.

In order to obviate this difficulty the American Pediatric Society has adopted a provisional nomenclature of the diseases of the mouth which was prepared by Dr. Forchheimer, of Cincinnati, and myself. To Dr. Forchheimer's extended investigations on this subject I am much indebted.

Diseases of the mouth occur more frequently and in much greater variety in infancy and in early childhood than at any later period of life. This depends partly on the anatomical conditions at different periods of development and partly on the external influences which are brought to bear upon the buccal mucous membrane. During the first three or four months of life the function of the salivary glands is not developed, and the flow of saliva is insignificant. This lack of saliva allows the mucous membrane of the mouth to be dry in comparison with that of the older subject. Even after the saliva is secreted the infant is more apt under certain conditions to let it flow from the mouth than to swallow it, so that the mucous membrane of the lips and mouth may present a different appearance in young infants, when they are attacked by various morbid processes, from that seen at a later period of development. We must also remember that the salivary glands in addition to their especial function are excretory organs, and that substances which are absorbed by the stomach may be eliminated by the mouth and in this way become sources of irritation and disease in the latter. The mucous membrane of the mouth during almost the whole period of infancy is subject to external sources of irritation to which older children, as a rule, are not liable. Thus, during the first year the mucous membrane is subjected to more or less mechanical irritation through the mechanism of sucking. At this period, also, it is very common for foreign organisms to be introduced into the mouth by means of the fingers either of the infant itself or of its attendant. It is not surprising, therefore, that we should meet with a great variety of pathological conditions in the mouth in infancy.

The organisms which occur in the mouth are so numerous that very few of them have as yet been differentiated in such a way that they can be known as the cause of the specific disease in which they are often found. We cannot, therefore, at the present time describe the various diseases of the mouth under their proper etiological headings, and we are forced to adopt provisionally the name of the pathological lesion which exists in them.

In almost every disease of the mouth which occurs in infants and in young children there is a coexisting inflammation of the mucous membrane. This inflammation may at times be very mild and often difficult to detect as such, but it still presents a recognizable pathological condition. This inflammatory condition, although not necessarily preceding the various diseases, yet in a large number of cases either exists as a basis on which the disease develops, or so closely accompanies it that the general name *stomatitis* (inflammation of the mucous membrane of the mouth) seems to be a proper term to use in connection with all these diseases.

Under the general heading stomatitis we can speak of most of the important diseases which affect the mucous membrane of the mouth in infancy and early childhood. These diseases may be divided into four general headings, according to the character of the lesions which occur in them.

The following table represents the provisional nomenclature of diseases of the mouth adopted by the American Pediatric Society :

TABLE 69.

STOMATITIS	Catarrhalis	Simplex.	
		Exanthematica	Secondary to the Exanthemata.
		Traumatica	Mechanical. Thermal. Chemical.
	Herpetica	Aphthosa.	
	Ulcerosa	Scorbutus.	
		Mineral Poisons	Arsenic. Lead. Mercury.
		And other diseases.	
	Mycetogenetica	Hyphomycetica	Thrush.
		Pseudo-Membranosa	Diphtheria. Tuberculosis. Syphilis, and like diseases.
		Gangrænosa	Noma

In accordance with this table the four general names which cover all these diseases are *stomatitis catarrhalis*, *stomatitis herpetica*, *stomatitis ulcerosa*, and *stomatitis mycetogenetica*.

STOMATITIS CATARRHALIS.

Stomatitis Simplex.—The form of stomatitis catarrhalis which is called the *simple* or *erythematous* form is commonly seen in young infants as a hyperæmic condition of the blood-vessels, causing diffuse redness of the whole buccal mucous membrane. This erythematous form is so common and so entirely without clinical significance that it may be considered as physiological and need only be referred to.

Stomatitis Exanthematica.—The second form, which is called *exanthematica*, is the condition of the mucous membrane which occurs secondarily to the exanthemata, and has already been described in connection with these diseases. It therefore need not be spoken of again.

Stomatitis Traumatica.—The third form, which is called *traumatica*, is the one which represents the characteristic stomatitis catarrhalis. The causes of the traumatic form of stomatitis catarrhalis are very numerous. They may be *mechanical*, *thermal*, or *chemical*.

Mechanical.—Among the most common mechanical causes may be cited the irritation produced by rubber nipples, too vigorous cleansing of the mouth, injudicious rubbing of the gums during dentition, and local irritation from a tooth.

Thermal.—The thermal form of traumatism may result from the administration of food which is too hot.

Chemical.—The chemical irritation may arise in various ways, as from lack of cleanliness in the mouth, with its resulting fermentation, and from the elimination of irritating products from the glands apparently connected in some way with disturbance in the gastro-enteric tract.

It is probable also that various forms of bacteria or their products may cause both mechanical and chemical irritation of the buccal mucous membrane. Our knowledge of the bacteriology of the mouth is as yet, however, so limited that we can scarcely undertake to describe the relation between special forms of bacteria and special lesions of the mucous membrane.

Pathology and Symptoms.—As the lesions which are seen in the mouth of an infant with stomatitis catarrhalis during life almost entirely disappear at death, and as very few post-mortem examinations have been made of these lesions, we can speak of the pathology and symptoms of this disease together.

The lesion is essentially an inflammatory one, and occurs in different grades. On examining the mucous membrane in these cases it is seen that the entire lining of the mouth is intensely reddened, that the temperature of the mouth is increased, that there is usually a certain amount of swelling, and that, although the mucous membrane may be dry under certain circumstances, especially at first, yet, as a rule, there is later a hypersecretion of mucus and saliva. The blood-vessels are so distended and their walls so weak that the slightest traumatism may cause their rupture, and the saliva is frequently mixed with a little blood. In older children the mucous membrane may be considerably swollen, especially behind the incisor teeth. In addition to this general condition of the mucous membrane of the mouth, at times the lips are found to be swollen and much reddened. The surface of the mucous membrane shows a number of small round prominences, which are the muciparous follicles. If complete occlusion of the ducts of these follicles occurs, great dilatation of the gland will take place, and a cyst may be formed. This, however, is a comparatively rare complication. In connection with the disturbance of the glands in the mouth the lymphatic glands are usually involved secondarily.

When the catarrhal condition is at its height the mucous membrane is so vulnerable that even slight traumatisms may cause abrasions. The most marked symptom of stomatitis is pain. The infant is restless, usually has a heightened temperature, and refuses to take its nourishment. The saliva is acid in its reaction, and when secreted in large quantities flows out of the mouth upon the chin and neck, sometimes causing considerable irritation. The tongue is dry and white at first, then becomes of a grayish color, and as the secretion of saliva increases the coating of the tongue is washed off and its surface becomes red.

Prognosis.—The prognosis of stomatitis catarrhalis is, as a rule, good. Although the disease does not run a definite course, yet in most cases after a few days the pathological condition improves and the symptoms grow less severe. The course of the disease is, however, often lengthened by the secondary conditions which arise from the gastric disturbances, which may be caused by swallowing the irritating secretions of the mouth. In weak, poorly nourished infants who refuse to nurse or to take the food which is given them, serious results may arise from a lack of sufficient nourishment, so that in these cases the prognosis is always grave. In older children the prognosis is very good.

Treatment.—Although stomatitis catarrhalis may run a favorable course without any treatment whatever, yet there are so many causes which may prolong its course or give rise to secondary affections that it is exceedingly important to treat the disease at once. The indications for treatment are to relieve the pain and to allay the irritation of the mucous membrane so that a sufficient amount of nourishment may be taken by the infant to prevent it from being harmed by a lack of nourishment or by a secondary disturbance of the gastro-enteric tract. If the cause can be ascertained, it should be removed at once. The local application of a one to two per cent. cold solution of bicarbonate or borate of sodium in distilled water is indicated. This solution should be used very gently every half-hour when the infant is awake, by means of a dropper, and occasionally on a clean swab of absorbent cotton. The infant should be systematically fed at regular intervals, whether it resists or not; and if it is not being nursed or will not suck from the nipple, a carefully modified milk at a temperature of about 32.2° C. (90° F.) should be administered with a spoon or dropper. There is no necessity for giving any drug internally in this disease.

When the stomatitis proves to be intractable and lasts for more than three or four days, the mouth can be gently touched with a cotton swab wet with a one per cent. solution of nitrate of silver. This should be done once a day, and the mouth washed carefully with cold sterilized water after the application.

When there are any abrasions which show a tendency to extend or to form an ulcer, they should be touched with a little nitrate of silver melted on the end of a silver probe. These abrasions are often so painful that in themselves they may prevent the child from taking its food, and after they have been treated with the nitrate of silver the child will often again take its nourishment readily. The following case represents stomatitis catarrhalis:

An infant, six months old, was reported to have been always healthy, and to have been nursed by its mother. It cut its first tooth, a middle lower incisor, when it was five months old. Nothing abnormal was noticed about the infant until it became fretful, restless, had a heightened temperature of about 38.8° C. (102° F.), and vomited occasionally. Although it did not cry a great deal, it frequently whimpered, as though in pain, and kept putting its fingers to its mouth. A few days later it refused to nurse. When it was put to the breast it appeared to be hungry and would take hold of the nipple vigorously, but immediately afterwards would draw its head away, as though sucking the nipple caused pain.

A physical examination showed nothing abnormal except in the mouth. The mucous membrane of the mouth, tongue, and gums was reddened, and small raised spots were seen corresponding to the positions of the muciparous glands. The mucous membrane of the tongue and lips was somewhat swollen and hot, and evidently sensitive to the touch. Where the tooth touched the tongue the inflammatory condition was especially marked, and it was possible that the sharp edge of the tooth was the original starting-point of the general inflammation.

In the above case the indications were for active treatment, as the infant was losing in weight from lack of sufficient nourishment. When the mouth is in this condition there is also a great liability to other diseases being implanted upon it, as the mucous membrane is very vulnerable when a pronounced stomatitis catarrhalis is present. The saliva flows from the mouth in such quantities and is so irritating that an eczematous condition is likely to be produced on the chin. The following prescription will be found useful in such cases:

PRESCRIPTION 82.

Metric.	Gramma.		*Apothecary.*	
℞ Sodii boratis	1	8	℞ Sodii boratis	gr. xxx;
Glycerini	7	5	Glycerini	ʒ ii;
Aq. destil. ad	120	00	Aq. destil. ad	℥ iv.
M.			M.	

This should be applied every hour while the child is awake. The chin should frequently be dried gently and a little vaseline applied if there is any eczema. If the infant is unwilling to nurse because of the stomatitis, the milk should be given by means of a dropper regularly every two hours until it will again take the breast. Improvement should be expected within four or five days.

STOMATITIS HERPETICA.

The name *herpetica* has been adopted for the next form of stomatitis, because it seems to represent most nearly the lesion which is seen on the mucous membrane, although it is not definitely settled that it is a true herpes.

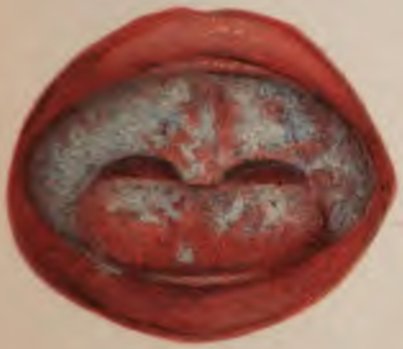
The disease consists of a catarrhal stomatitis in the course of which certain lesions resembling sub-epithelial vesicles surrounded by areolæ occur irregularly and in different parts of the entire buccal cavity. This form of stomatitis has usually been known as stomatitis aphthosa (*ἄφθα*, an eruption or ulceration). This name was given to it by Bohn as distinctive from the other forms of stomatitis, but it does not represent the affection especially well.

ETIOLOGY.—As a rule, when the mucous membrane of the infant's mouth is in a normal condition it is not readily affected by the various irritants which produce its special diseases. When a catarrhal condition is present the mucous membrane becomes more vulnerable and the various diseases have an opportunity to develop. This apparently is illustrated in the case of stomatitis herpetica, in conjunction with which affection a catarrhal stomatitis is always found. No cause, either local or general, has as yet been determined for this disease. Various micro-organisms have been observed in the mouth when it is affected by stomatitis herpetica, but no actual connection has been discovered between them and the disease. This affection may be found associated with a number of other diseases, but usually occurs alone. It does not seem to be contagious, nor to be especially connected with diseases of the gastro-enteric tract or with dentition, although it very commonly occurs during the dental period. It appears to be the result of certain deleterious influences which act upon the nerve-centres and produce an herpetic efflorescence on the mucous membrane which corresponds closely to that which is seen in herpes on the skin.

PATHOLOGY AND SYMPTOMS.—In addition to the usual lesions of a stomatitis catarrhalis, spots, not necessarily symmetrical or unilateral, of different sizes and of different shades of white or grayish white, appear in various parts of the mouth, especially on the inner surface of the lip, on the side and under surface of the tongue, and on the gums. These lesions do not affect the follicles of the mouth, and the efflorescence cannot be called follicular, as it is closely connected with the muciparous glands. The lesions make their appearance with great rapidity, and develop very

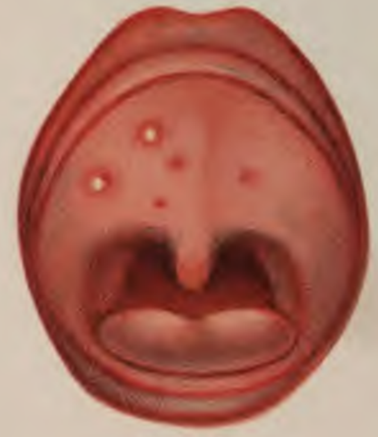
PLATE XI.

1.



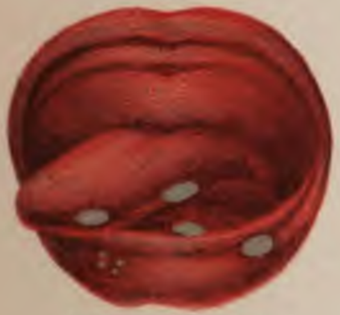
Thrush

2



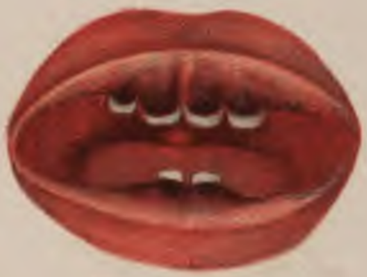
Varicella.

3.



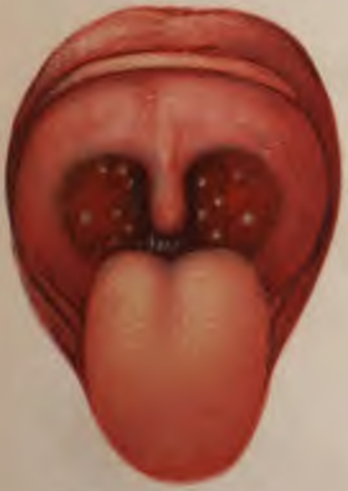
Stomatitis Herpetica.
(Aphthosa.)

4.



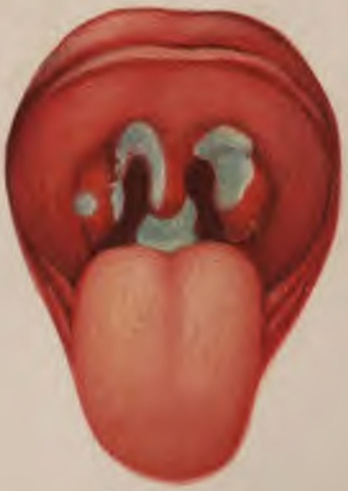
Stomatitis Ulcerosa.
(Scorbutus.)

5.



Follicular Tonsillitis.

6.



Diphtheria.

quickly from a macule into what is supposed to be a vesicle. The action of the secretions of the mouth upon these lesions necessarily prevents them from having the same definite appearance that they would present on the skin. The course of the disease so strongly simulates that of herpes that at present it would seem wise to consider the efflorescence herpetic.

The general appearance of the efflorescence when at its height is that of a sub-epithelial vesicle, somewhat glistening, of a whitish-gray color, and surrounded by a red areola. The lesions may be only a few in number, scattered irregularly over the parts of the mucous membrane which I have already described. At times, however, the efflorescence is very diffuse, sometimes appearing as minute grayish points, which may become much larger and cover the mucous membrane so thickly as almost to simulate a false membrane. In a still later stage of the disease these lesions may break down and form small superficial ulcers.

An infant or young child affected by stomatitis herpetica presents a very characteristic appearance. It looks dull and apathetic, and wishes to lie quietly in bed. It usually has a heightened temperature, and evidently suffers from pain and heat in its mouth. The saliva flows from the mouth in large quantities, and often irritates the chin and neck to such an extent that an eczematous condition results. The child refuses to take its nourishment, and is very fretful and restless. These symptoms continue for four or five days or a week, and sometimes extend over a period of two weeks, the disease then disappearing of itself; in fact, it appears to be self-limited. Unless the lesions of stomatitis herpetica are complicated by those of stomatitis ulcerosa, the saliva is never fetid.

PROGNOSIS.—The prognosis of stomatitis herpetica is very favorable, although infection from other diseases may take place. This latter occurrence is, however, exceedingly rare. Relapses are very uncommon in this form of stomatitis, and the lesions usually heal readily.

TREATMENT.—There is no internal treatment which is of benefit in this disease. The indications for treatment are to allay the irritation of the mucous membrane and to prevent its infection by some other poison. The mouth in general should be treated as has just been recommended in the case of stomatitis catarrhalis. As a rule, very little treatment is necessary beyond occasionally cleansing the mouth with the solution (Prescription 82, page 619) already mentioned. The ulcers which do not heal readily can be touched with nitrate of silver. The feeding should be at regular intervals and if necessary forced.

Plate XI., facing page 620, represents stomatitis herpetica in a boy four years old. He was perfectly well until two days previous to the appearance shown in the plate; he then began to be feverish, was restless at night, refused to take his food, and seemed quite sick. On the following day the entire mucous membrane of the mouth was found to be affected with stomatitis catarrhalis, and somewhat later the herpetic form of stomatitis, the lesions of which are shown in the plate.

On drawing down the lower lip on the right side a number of small grayish-white spots surrounded by a somewhat deeper, reddened mucous membrane were seen. At a little distance from them, on the left side of the lip, close to the gum, was apparently a sub-epithelial vesicle. On the inner side of the lower gum one of these vesicles had broken down, and a small superficial ulcer covered with a grayish-white exudation was seen. There were also lesions of the same vesicular character along the left edge of the tongue. The entire mucous membrane of the mouth was intensely reddened, and the case illustrated stomatitis catarrhalis as well as stomatitis herpetica.

The child absolutely refused to take food, and so it had to be forced upon him. In a few days the more severe stage of the disease passed away and he took his food. The treatment was to bathe the inflamed mucous membrane with cold sterilized water, and small quantities of an alkaline modified milk were given to him. As this child appeared lying with his eyes half closed, with flushed cheeks, in an apathetic condition, occasionally whimpering as if in pain, and with the saliva flowing continuously from his mouth on the pillow, one could readily diagnosticate the disease stomatitis. When, in addition, the characteristic lesions of the mucous membrane irregularly distributed throughout the buccal cavity are noticed, and no evidence of a membranous exudation can be found, there need be no doubt as to the diagnosis. Internal remedies are not needed in a case of this kind. Chlorate of potassium, which is so commonly used in all diseases of the mouth, is not indicated in the forms of stomatitis which have just been described.

In connection with this form of stomatitis may be mentioned certain lesions occurring in the mouths of new-born infants which have been called *Bednar's aphthæ*. These lesions consist of small superficial ulcers usually having a grayish coating, and appearing on the posterior part of the hard palate and on the soft palate. They are now supposed not to represent a specific disease, but to be the result of traumatism, such as may arise from a badly-shaped rubber nipple or from undue violence in washing the mouth.

They are to be treated as any local irritations of the mouth should be,—namely, by removing the cause, applying a solution of bicarbonate of sodium, and, if necessary, touching them with nitrate of silver.

STOMATITIS ULCEROSA.

By stomatitis ulcerosa we mean a peculiar pathological process of the mucous membrane of the mouth occurring only when there are teeth and affecting the gums around the teeth.

ETIOLOGY.—This affection of the mouth may occur in the course of a number of diseases, notably in scorbutus. It may also be produced by the internal administration of such mineral poisons as arsenic, lead, or mercury. Occasionally it may occur as a local affection without known cause, but it is probably produced by the irritation of some form of micro-organism not yet determined, although the pyogenic bacteria are very commonly present.

The most common form of stomatitis ulcerosa produced by the mineral poisons is that which is seen in connection with mercurial salivation.

As in the other forms of stomatitis, it is probable that the mucous membrane is first affected by a catarrhal process which renders it vulnerable to the special irritation which produces stomatitis ulcerosa. This preceding stomatitis catarrhalis may be produced directly by local irritation in the mouth itself, or may be the result of some disturbance of the general system. For this reason stomatitis ulcerosa, as a rule, does not affect primarily a healthy individual. Thus, a poorly nourished child, and one whose mouth is not properly cared for, will be more apt to have this disease develop than one who is correctly fed and whose mouth is clean.

PATHOLOGY.—The pathological condition is one of necrobiosis; that is, there is softening as well as death of the tissues. The disease, although starting in the mucous membrane, may extend to the periosteum, and even produce necrosis of the bone. It begins at the free border of the gums, and can extend in all directions, but it never passes beyond the mucous membrane of the mouth. The softening of the tissue not only changes its consistency but also renders it more movable, and in this way the gums at times become so swollen and loosened that they may entirely cover the teeth.

SYMPTOMS.—Stomatitis ulcerosa is usually preceded by moderate constitutional symptoms, such as fever, loss of appetite, and fretfulness. The mucous membrane of the gums at the free margin of the teeth becomes reddened and soon begins to swell. The normal curve of the gum becomes almost a straight line and covers the lower part of the teeth. The gums in the space between the teeth remain unaltered at first. The mucous membrane then begins to change in color and becomes purplish. Extreme congestion and softening of the tissues allow hemorrhage to take place from the slightest pressure. Although the anterior surface of the gums is most commonly affected, yet in severe cases the posterior surface is also involved. As the process develops further the gum becomes more and more loosened as it extends over the teeth. A mucopurulent secretion collects between the gums and the teeth and causes a fetid odor. According to Forchheimer, a yellowish seam then appears at the top of the swollen outline of the gum. This is due to the molecular destruction which has already begun. This seam is at first very narrow, but later it may become broader and involve almost the whole of the gum. In connection with this characteristic appearance of the gums there is a great hypersecretion of saliva. At the height of the disease the child evidently suffers from pain in the mouth, cries a great deal, and rapidly emaciates. The lymphatic glands are usually swollen, and remain so until the disease has ended. When the yellowish material which constitutes the seam already referred to is removed, an ulcerated surface will be found beneath. Although stomatitis ulcerosa may begin about any of the teeth, its most common starting-point is around the lower incisors. As the disease improves, the gums gradually become less swollen and congested, returning to their normal relation to the roots of the teeth, and the salivation disappears.

Diagnosis.—The differential diagnosis of stomatitis ulcerosa, when the lesions of the disease are marked, presents no difficulty. Although an herpetic efflorescence may occur coincidently with the ulcerative form, yet the pictures of the two diseases are so different that we know at once that we are dealing with two affections rather than with one. There is no other disease of the mouth in which the gums assume the purplish hue and the swollen, soft, and loosened condition which are characteristic of stomatitis ulcerosa.

Prognosis.—The prognosis of stomatitis ulcerosa depends upon its cause and whether it is treated or not. The tendency is, however, after a variable period of discomfort to the child, for the disease to disappear.

If the affection is the result of one of the constitutional diseases, such as syphilis or scorbutus, it disappears if the treatment of the specific disease is beneficial, otherwise it continues, and may finally lead to death by exhaustion.

Treatment.—The local form of the disease is best treated by the internal administration of chlorate of potassium or by this drug in solution used as a wash for the mouth. Chlorate of potassium must, however, be given with great precaution to infants and children, as in certain cases it acts as a poison, some infants being affected by even minute doses. The symptoms which show that chlorate of potassium is producing deleterious effects in infants who are most likely to be affected by the drug are drowsiness and suppression of urine, with weakness of the heart and sometimes cyanosis. When these symptoms follow the administration of the drug, it should be omitted at once and a simple wash of borate of sodium used. Chlorate of potassium when given internally has been found to be secreted in the saliva within five or ten minutes, and thus has an opportunity of producing a direct effect upon the lesions of the gums. The doses of chlorate of potassium, which, it has been found, can be safely administered to infants and children, should be remembered when prescribing the drug. Table 70 gives the minimum doses which can safely be taken in the twenty-four hours at different ages, and which are sufficient to produce the specific effect of the drug in treating cases of stomatitis ulcerosa.

TABLE 70.

Amount of Chlorate of Potassium which can be safely given in Twenty-four Hours at Different Ages.

Age.	Gramme.	
Under 1 year	1	0
1 to 2 years	1	5
2 to 6 years	2	0
6 to 8 years	2	5
8 to 14 years	3	0

In order that the chlorate of potassium shall produce the best effects it should be given frequently. The total amount for twenty-four hours which is to be given at any special age is to be placed in a tumbler and dissolved in as many tablespoonfuls of sterilized water as there are doses to be given within the twenty-four hours. Let us suppose that the number of hours which the child sleeps is ten: then fourteen tablespoonfuls of the solution should be prepared, and the child should be given one tablespoonful every hour when it is awake. The administration of chlorate of potassium at first usually produces considerable smarting and pain in the mouth as it passes over the inflamed surface of the mucous membrane. These symptoms, however, last for only a short time, usually disappearing entirely after thirty-six to forty-eight hours.

Under this treatment the disease is ordinarily cured in a week or ten days. The treatment should, however, be continued for a number of days after the mouth is apparently entirely well.

When deeper ulceration has taken place, its disappearance may sometimes be expedited by the application of nitrate of silver. When a sequestrum has formed, it must be removed. Frequent washing of the mouth with sterilized water administered by means of a dropper is also very important, especially after the taking of food. An alkaline diet is indicated.

Plate XI., facing page 620, represents a case of stomatitis ulcerosa in an infant ten months old, in whose mouth were shown the characteristic lesions of the disease. In this case the disease happened to be secondary to scorbutus, the affection for which the infant was treated.

The infant had six teeth, and the mucous membrane was affected only at the junction of the gums with the free surface of the teeth. The other parts of the mucous membrane of the mouth were reddened, but not markedly so. The portions of the gums affected were swollen, purplish, loosened, and almost covered the teeth. There was a considerable flow of saliva, with a fetid odor from the mouth. An appearance of this kind is diagnostic of stomatitis ulcerŏsa.

The following case of stomatitis ulcerosa was apparently of local origin:

A girl, three and a half years old, had always been healthy, and had had no diseases of any kind. She began to have loss of appetite, a temperature varying from 38.3° to 39.4° C. (101° to 103° F.), and to be very fretful. Three days later the gums were noticed to be swollen, to be of a dark-red color, and her breath had a fetid odor. She was rather apathetic and wished to remain in bed. There was considerable salivation. During the first three days her restlessness was so excessive at night that 0.3 gramme (5 grains) of bromide of potassium had to be given to her to produce sleep.

In two or three days more the disease ran its course, and entire recovery took place.

STOMATITIS MYCETOGENETICA.

There are three forms of vegetable parasites which occur in or upon the human body: (1) bacteria, or fission-fungi (schizomycetes); (2) yeasts, or yeast-fungi (saccharomycetes); (3) moulds, or mould-fungi (hyphomycetes). The changes in the tissues which are due to fungi are termed *mycetogenetic metamorphosis*, and thus the pathological conditions in the mouth which are produced by any of these forms of fungi may be designated by the general term *mycetogenetica*. Under this general heading of mycetogenetica we can include the various forms of stomatitis which are caused by fungi.

Stomatitis Hyphomycetica (*thrush*).—ETIOLOGY.—The disease which is commonly called thrush is produced by a fungus which finds its nidus upon the surface of the mucous membrane of the mouth, usually in young infants. This fungus was formerly supposed to be the *oïdium albicans*, but it is now known not to be this organism, and the precise form of mould which it represents has not yet been determined. We merely know that this growth of thrush is one of the mould-fungi, and we can therefore at present only classify it as *stomatitis hyphomycetica*.

The moulds are complex in their structure, and as commonly described consist of a series of delicate jointed threads (*mycelium*) in which spores are developed. Hyphomycetic growth is characterized by having the spores naked on conspicuous threads. The fungus of thrush may be found on any of the mucous membranes of the body. It has also been found in various organs, as in the brain and the lungs, and from the surface of ulcers it has on rare occasions penetrated the blood-vessels and given rise to visceral metastases. The usual place for it to appear, however, is the mucous membrane of the mouth. It is a local disease, and may occur in the mouths of healthy children as well as in those who are diseased. It is more likely, however, to be ingrafted upon a diseased than upon a healthy mucous membrane, in accordance with the rule which I have already stated. A catarrhal condition of the mucous membrane, by displacing the epithelial cells and thus interfering with their protection of the mucous membrane, affords the readiest means for the development of the fungus of thrush. It is therefore more likely to be found in the mouths of children who are suffering from various diseases or who are ill cared for. It may be carried to the mouth in various ways, either on dirty nipples or by the finger.

PATHOLOGY.—The growth may take place on both squamous and cylindrical epithelium. According to Forchheimer, the first lodgement of the fungus comes between the epithelial cells of the mouth, and from this the growth works its way under the free surface of the mucous membrane. When directly on the free surface the growth is not so luxuriant and is principally in the mycelium form. In the case of a mucous membrane lined by flat or squamous epithelium, the growth is facilitated by the relation of the cells to one another. In a membrane lined by cylindrical epithelium, the growth takes place, but not so readily, because there is but one layer of cells. After the first development the growth goes on very rapidly, and after it has found a nidus the cells are pushed aside and are surrounded by mycelium, the whole presenting the characteristic appearance of thrush. The growth begins in small spots, sometimes one, sometimes more, and at times the entire surface of the mucous membrane is covered with it. The fungus develops within the epithelium, and it requires considerable rubbing to remove the growth.

SYMPTOMS.—An attack of thrush usually begins with local symptoms of catarrhal stomatitis. At times, however, no symptoms are present, the fungus being the first abnormal condition which is noticed. The appearance of the fungus resembles closely that of curdled milk, though it is often of a rather grayish color. It does not look like a membranous exudation, but is raised in small patches above the level of the mucous membrane. The fungus usually develops on the inner borders of the lips, on the gums, on the tongue, and on the hard and the soft palate. It may extend to the tonsils and pharynx, and even into the œsophagus. In the latter locality at times it has been found to grow so thickly that the lumen is almost entirely occluded. The local symptoms are commonly those of a mild catarrhal stomatitis. The general symptoms depend upon the extent of the local disease from which the infant is suffering. Infants affected with this disease soon become atrophic, from a lack of proper nourishment, as they are often unwilling to take their food or cannot swallow it without difficulty.

DIAGNOSIS.—The differential diagnosis is seldom difficult to make. Curdled masses of milk on the inner surfaces of the lips and on the gums may resemble closely the fungus of thrush, but the former is easily wiped away, while the latter is difficult to dislodge. The disease is definitively determined by placing some of the growth under the microscope, where it presents characteristic appearances.

PROGNOSIS.—The prognosis of thrush varies according to the general condition, the vitality, and the age of the subject on whom it is engrafted. The disease may last indefinitely if the mouth is not carefully treated, and its prolongation may render the prognosis more grave. When the growth is very extensive, as in the cases in which it has invaded the œsophagus, the prognosis is very unfavorable. In these cases disturbances of the gastro-enteric tract are apt to arise and to increase the likelihood of a fatal issue. As a rule, however, if the infant's health can be maintained, and if the local treatment is carried out thoroughly, the prognosis is favorable.

TREATMENT.—The treatment should be directed to the local care of the mouth and to supporting the strength by proper nourishment and stimulants until the fungus has been eradicated. Care should be taken that everything connected with the infant, especially the nipples and bottles from which it is to be fed, should be aseptic, so that it shall not be continually reinfected or infect other children. The mouth after each feeding, and also between the feedings, should be thoroughly and somewhat vigorously rubbed with the solution which has already been recommended in the treatment of stomatitis catarrhalis.

When the disease is in the œsophagus it is best treated by the introduction of a soft rubber tube, in order that the growth may thus be mechanically separated from the mucous membrane.

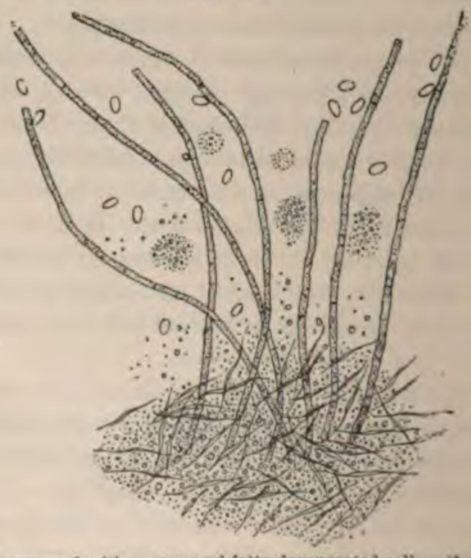
In many cases the disease is very intractable. No special drug appears to be of use in these cases, and they can be cured only by the unremitting and patient removal of the growth as just described.

Plate XI., facing page 620, Thrush, represents the mouth of an infant three months old, who refused to take the bottle for a month, was emaciated and fretful, and at times vomited.

A careful physical examination failed to detect anything abnormal except in the infant's mouth. On gently depressing the tongue and lower jaw, it was seen that the soft and the hard palate, the tongue, the gums, and the inner surface of the lips were covered almost entirely with white and grayish-white masses, in texture somewhat resembling curdled milk, and rising above the level of the epithelium. Between these patches the mucous membrane was reddened. There was a moderate flow of saliva. This morbid growth apparently did not extend into the pharynx. The patches could not be removed readily, as would be the case if it were curdled milk, and the growth evidently passed between the epithelial cells down to the underlying mucous membrane, where it was held so closely that it required considerable rubbing to separate it. In this case the growth was so extensive that it simulated a membrane in some places, but its generally roughened surface, its elevation above the level of the mucous membrane, and the characteristic appearances in other parts of the mouth rendered its recognition quite simple.

On placing some particles of this growth in glycerin under the microscope a tangled mass of fine, almost translucent, membered threads were seen as represented in Fig. 130.

FIG. 130.

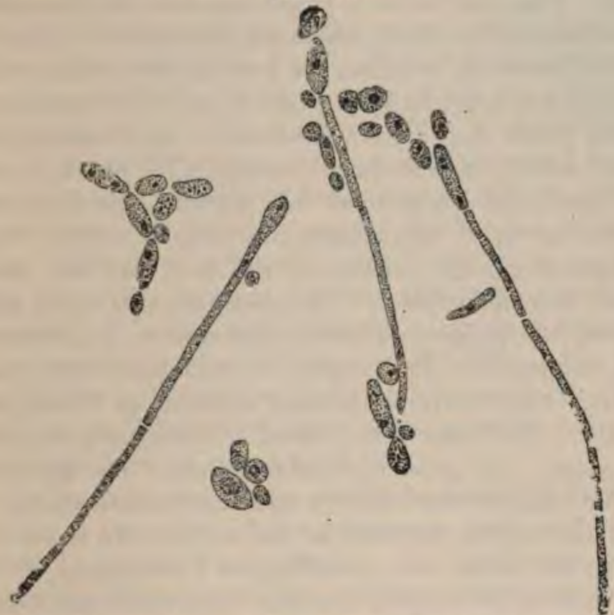


Mycelium of thrush interspersed with spores and fatty degenerated cells. (Low power Zeiss Oc. 3. Objective DD, glycerin.)

Interspersed among these threads were bright, glistening, oval bodies, which were the formed spores, and also fatty degenerated cells and fine detritus. This combination of appearances represents the pathological processes which we find in thrush.

Fig. 131 represents some shreds from the same specimen, but much more highly magnified. In this specimen can be seen the formation of the spores in the mycelium.

FIG. 131.



Thrush showing the formation of spores in the mycelium. (Zeiss Oc. 3, homogen. immer. 2.0 mm.)

Stomatitis Pseudo-Membranosa.—Under this heading I shall merely refer to those pseudo-membranous conditions which occur in diphtheria, tuberculosis, syphilis, and diseases of a like class. The former two are so rarely seen in the mucous membrane of the mouth that it is not necessary to describe them. The lesions which occur in the mouth in syphilis have already been described when speaking of that disease.

Stomatitis Gangrænosa (*noma, cancrum oris*).—Stomatitis gangrænosa is the rarest and most fatal form of stomatitis which occurs in children. It is usually met with between the ages of three and seven years. It is a disease characterized by a gangrenous process which begins on the gums or on the inner surface of the cheek and spreads with great rapidity to the adjoining tissues, all of which can be involved and quickly destroyed

ETIOLOGY.—It is probable that there is a specific germ which causes this disease. This organism has, however, not yet been determined. It is supposed that it does not attack a healthy mucous membrane, and that one of the other forms of stomatitis, especially stomatitis catarrhalis, and in some cases stomatitis ulcerosa, precedes it. Furthermore, stomatitis gangrænosa seldom attacks healthy children, but usually affects those who have other diseases and are greatly debilitated. It occurs most commonly secondarily to the acute exanthemata, especially measles. The disease is also said to result from the administration of mercury in too large doses.

It begins as a reddened, hard spot in the mucous membrane, usually of the cheek. This soon becomes gangrenous and extends rapidly through the entire thickness of the cheek, producing perforation. It may also extend laterally in all directions, attacking the bone as well as the other tissues.

SYMPTOMS.—The first symptom which is apt to be noticed is the gangrenous odor which comes from the mouth. On examination an ulcer will be found which tends to spread rapidly. The cheek becomes much swollen, is hard and œdematous, the œdema especially affecting the tissues under the eye. The gangrenous process extends very rapidly, at times destroying large portions of the face, and also involving the bones, which become denuded. The teeth become loose and fall out. The odor from the gangrenous tissue is excessive. The flow of saliva is very much increased. The degree of suffering which the children undergo varies very much; sometimes it seems as if they suffered no pain whatever. The temperature varies, at times being raised and again being subnormal. The pulse is weak and rapid. The appetite is diminished, and the children are likely to have diarrhœa, probably due to the infectious nature of the products of the mouth which are swallowed. Hemorrhages are rather rare, according to Forchheimer, as the blood-vessels are usually filled with thrombi. Secondary affections, such as catarrhal pneumonia from the inhalation of septic material, are not uncommon. The child may die from one of these secondary affections, or it may become more and more weakened by the local condition, and unless the morbid process is arrested it will die eventually from exhaustion.

DIAGNOSIS.—The diagnosis of this disease, except in its earlier stages, is not difficult. At times, however, a local ulcerative process produced by a decayed tooth may simulate closely stomatitis gangrænosa. In these cases the diagnosis is made more difficult by the fact that the tissues of the cheek may become hard and look as though perforation might take place. Coincidently with this condition the ulceration of the gum and often of the mucous membrane of the cheek, with the foul odor which emanates from it, makes the similarity of the two diseases very striking. In simple ulceration from a tooth, however, active local treatment with solutions of myrrh or of soda combined with frequent washing of the mouth with sterilized water is soon followed by marked improvement, while when stomatitis gangrænosa is present the morbid process continues to extend.

PROGNOSIS.—The prognosis in cases of stomatitis gangrænosa which are untreated is almost universally fatal. Cases have been known, however, in which a line of demarcation has formed around the gangrenous spot, granulations have arisen, and cicatrization has followed, leaving extensive scars. If the disease is treated by extirpation of the diseased tissues in the very beginning, the prognosis becomes more favorable. When the disease has perforated the cheek and the gangrenous process has become extensive, the child is seldom relieved even by surgical treatment.

TREATMENT.—Care should be taken when a child is affected with a disease of an exhausting nature that its mouth is kept thoroughly cleansed, for we can never tell when or in what individual the mucous membrane may become vulnerable to the organism which produces stomatitis gangrænosa. In stomatitis gangrænosa it is very important for the success of the treatment that it should be begun very early in the disease. When the diagnosis has been definitely made, it is wiser not to temporize with applications of nitrate of silver and other drugs, but at once to place the case in the hands of a surgeon and have the entire area of the invaded tissues excised. It is also well after the gangrenous process has been removed by the knife to destroy an area of healthy tissue by means of the Paquelin thermo-cautery or by the galvano-cautery. There should be no delay in operating upon these cases, as great destruction of the tissues may take place in even a few hours.

After the operation the tissues should be inspected frequently, to see whether there is any return of the gangrenous spots, and, if found, these spots should be removed immediately. As the disease is very apt to recur, plastic operations to obviate deformity should not be undertaken very early after the operation.

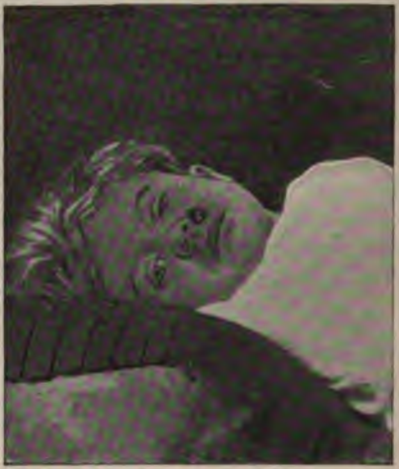
In treating these cases surgically it must be remembered that the child is in a very debilitated condition, and that if it is suffering from any especial disease treatment directed to that disease is indicated, also that stimulants are required to prevent the already weakened child from dying of exhaustion following the operation.

The following case, a girl four years old, represented in Fig. 132, was brought to the hospital to be operated on for stomatitis gangrænosa.

In this case the disease was apparently primary, and began on the left side of the mucous membrane of the mouth. It spread rapidly, and, although treated by local applications to the mouth of various solutions, had broken through the left cheek close to the ala nasi. The teeth were loose in the middle of the upper jaw, and there was a certain amount of alveolar necrosis. There was a strong gangrenous odor from the mouth and the tissues of the cheek, and a considerable flow of saliva. The child's general condition was fair, but she was becoming more debilitated, had lost her appetite, and had a slightly raised temperature.

The cheek was operated on the day after the child entered the hospital. The wound healed readily. One year later the child again returned to the hospital. The right cheek was found to be much swollen and indurated, especially under the right eye. The periosteum of the lower jaw on the right side was affected, and the necrotic process had undermined the whole cheek as far as the orbit. The child was again operated on without any external opening of the cheek. The wound healed, and the child was discharged from the hospital, but returned some months later with a spontaneous opening on the right cheek. This was again apparently cured by operation. Two months later the child was found to have in the lower jaw a process similar to that which had occurred in the upper jaw. Her health was poor, she was pale and weak and had loss of appetite. She was operated upon again, and a sequestrum was removed from the lower jaw. She then improved. Some months later she was apparently in fair health.

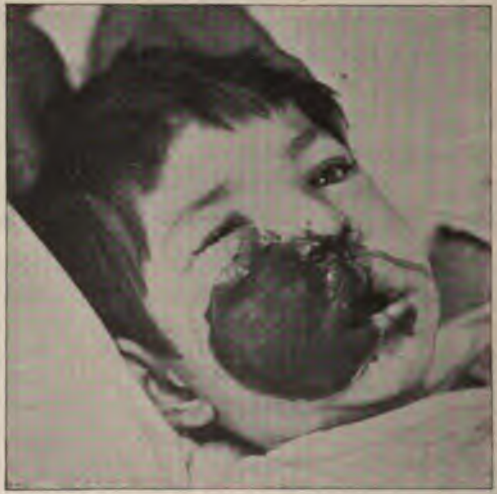
FIG. 132.



Stomatitis gangrænosa, left cheek. Female, 4 years old.

The microscopic examination of the gangrenous tissues removed at the operation presented nothing significant of any especial disease, and a culture showed only a few streptococci.

FIG. 133.



Stomatitis gangrænosa secondary to measles and pneumonia. Female, 5 years old.

The following case of stomatitis gangrænosa occurred in a girl five years old:

The disease was preceded by pertussis, measles, and a broncho-pneumonia. After she had had the pneumonia for seventeen days her right cheek began to swell and a bad odor came from her mouth, but nothing especial could be found in the mucous membrane of the buccal cavity. Four days later the swelling of the cheek had much increased, and there was œdema of the lips and eyelid so that the right eye was partly closed. The swelling was semi-fluctuating. The temperature varied from 38.3° to 39.4° C. (101° to 103° F.), and the cough had much lessened. On the following day a bluish-black spot about 1.5 cm. ($\frac{5}{8}$ inch) in circumference appeared at the right corner of the mouth, and this rapidly increased during the day. Two days later the dark-colored area had increased considerably in size and presented a circular outline with a clearly marked line of demarcation.

The child also had a profuse greenish diarrhœa. On the following day the dark area rapidly extended, and soon involved the whole of the right cheek, the right side of the mouth, and the right nostril. There was no external loss of tissue. The child was extremely emaciated, and from the beginning of the attack was in a hopeless condition, so that radical treatment of the disease was deemed inadvisable. It died suddenly on the following day. Fig. 133 represents the case.

GLOSSITIS.

Glossitis is so rare a disease in children that the possibility of its occurrence only need be mentioned. In this affection there is an acute inflammation of the tissues of the tongue, accompanied by fever, enlargement of the organ, and considerable pain. There is usually a hypersecretion of saliva, and at times the obstruction to respiration from the occlusion of the throat by the greatly enlarged tongue produces somewhat alarming symptoms, though, as a rule, not serious ones.

This disease may be caused by direct injury to the tongue from corrosive substances, by heat, or by the stings of animals, and sometimes probably by sepsis. It runs a variable course; it is not especially serious, and tends to recover after a few days. The treatment is purely symptomatic. The frequent local application of ice and of ice-cold alkaline solutions to the tongue and mouth is indicated.

LINGUA GEOGRAPHICA.

A condition of the dorsum of the tongue is sometimes met with, which, for want of a better name, is called *lingua geographica*, "mappy tongue," or "wandering rash." One or more small patches appear on the dorsum or side of the tongue, which in a few days may spread and coalesce, often covering a large portion of the surface. They diminish in size or fade with equal rapidity, to recur at variable periods. The patches are red and smooth, and the filiform papillæ are absent. The rest of the tongue appears normal, except that the papillæ on the borders of the denuded portions are white and prominent. The etiology of the disease is unknown. It occurs almost exclusively in children or in young adults who have been subject to it from childhood. It is very benign, and gives no discomfort to the child. Its principal importance lies in the fact that it is sometimes mistaken for a symptom of some more serious disease. No form of treatment has been found useful. It recurs periodically for months or years, but does not tend to increase in severity nor to lead to other diseases.

MICROGLOSSIA.

In some individuals an arrest of development of the tongue produces the condition called microglossia, in which the tongue is to a varying degree smaller than normal.

MACROGLOSSIA.

The opposite condition, macroglossia, in which the tongue is enlarged, is more common than microglossia. It is usually a congenital lesion, and is especially marked in cretins. The prominent feature of the affection is a prolapse of the tongue, which is often enormously enlarged in every direction, is usually of a deep violet color, and is covered with a thick, whitish coat. The protruded tongue is indented and even ulcerated by the teeth, which are often pushed forward and become carious. The saliva flows continuously from the mouth, the lower lip becomes thick and ulcerated, and the forcing forward of the lip, larynx, and velum palati by the weight of the tongue renders suction, mastication, and deglutition difficult. The nutrition of the child is thus much interfered with, and this interference is one of the most serious results of the disease. This condition is not a glossitis, but a deformity which seems to be associated with certain other malformations of the body. In these individuals the hands and feet are apt to be large, thick, and purplish.

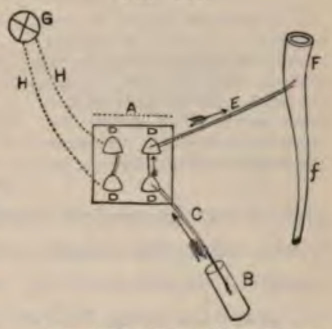
Macroglossia appears in two forms. One is the fibrous, in which the connective tissue is pathologically increased between the muscular fibres. The other is a cavernous cystoid degeneration of the interstitial connective tissue, by which the resulting spaces come in connection with the lymph-vessels, constituting a condition closely resembling cavernous angioma, from which it receives its name of *lymphangioma cavernosum.*

The disease seldom tends to recover, and the treatment is to give as much relief as possible to the great discomfort which arises from it, by cleansing the mouth frequently with alkaline solutions. Especial care should be directed to the nourishment of the child. In extreme cases surgical interference is indicated when the child's respiration and general nutrition are affected, and in some cases great improvement is accomplished by the removal of part of the tongue.

DIFFICULT DENTITION.

The normal development of the teeth in infancy and childhood is a physiological process. The teeth are developed at birth to a certain degree, and merely increase in size during infancy until they pierce the gums and assume their places in the mouth. In many cases the process of dentition gives rise to no morbid conditions whatever. The idea that dentition occasions the various diseases with which it was formerly supposed to be associated is an erroneous one. From the fourth or fifth month, however, until the completion of dentition in the latter part of infancy, various nervous disturbances are so closely associated with irritation in the mouth that in this sense dentition may be considered responsible for many of the slight ailments which arise at this period of life. The mouth at this time frequently becomes hot, and sometimes dry, although there may be a hypersecretion of saliva. There is evidently much discomfort in the region of the gums, as the infant is continually rubbing them with its fingers and seems to get relief from biting on hard substances. Such infants may become much prostrated and may lose their appetite, and thus their nutrition may be interfered with, without any discoverable cause for these abnormal conditions beyond the general nervous irritation which arises from the feeling of discomfort in the mouth and head. In the more extreme cases the infant will be so restless at night that it scarcely lies still for half an hour at a time, and may spend night after night crying out occasionally as though in pain, and knocking its head against the sides of its crib, so that in some cases the crib will have to be padded. These infants also have to be guarded sometimes from knocking their heads against the floor or wall, as they seem to become almost frantic from the continued irritation from which they are suffering. These symptoms occur with such regularity at a time when a tooth is in its final stage of development, and cease so uniformly when the tooth has attained its growth, that the causal relation between the tooth and these nervous symptoms seems more than probable. This rather indefinite clinical association of dentition and nervous symptoms is, however, partially explained by the analogous symptoms arising from the anatomical relationship which exists between the roots of the teeth and the ear. It has long been noticed that in certain infants, during the completion of the development of a tooth, symptoms connected with the ear will manifest themselves. These symptoms are usually produced by a congestion of the blood-vessels of the ear, which is accompanied by pain, and sometimes results in inflammation. They are evidently of reflex origin. Fig. 134 explains the influences which an irritation of some distant part of the economy may exert on the blood-vessels of the ear.

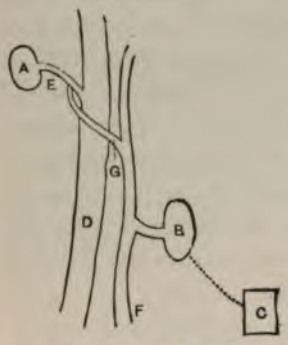
FIG. 134.



A, sympathetic ganglion; B, sensori-motor nerve; C, afferent sympathetic fibres from sheath of B; D, caudate cells; E, efferent sympathetic fibres proceeding to artery *f*; F, artery dilated; *f*, normal size of artery beyond the sympathetic influence; G, general vasomotor centre; H, H, the dotted lines indicating the course of the fibres forming the roots of the ganglion in the spinal cord to the general vasomotor centre G. (Woakes.)

The general vascular disturbance in the ear, represented either by an uncomfortable feeling of fulness or by general pain, may be produced in cases of difficult dentition by this close connection between the sensorimotor nerves and the sympathetic. According to Woakes, a considerable portion of the blood-supply of the membrane of the drum is derived from the artery that leaves the internal carotid in the carotid canal and proceeds by a very short course directly to its destination. Being thus closely connected with a large arterial trunk, this small tympanic branch is very favorably situated for a speedy augmentation of its blood-supply. The nervi vasorum constituting the carotid plexus at this part of its course come largely from the otic ganglion. On the other hand, the inferior dental nerve supplying the gums and the teeth also communicates with this ganglion.

FIG. 135.



A, tympanic cavity; B, otic ganglion; C, tooth; D, internal carotid; E, tympanic branch; F, auriculo-temporal nerve; G, auricular branch of auriculo-temporal nerve. The dotted line connecting B and C represents the inferior dental nerve.

We thus arrive at a direct channel of nerve communication between the source of irritation in the mouth and the vascular supply of the drum-head. The earache which arises in these cases is produced by the vessels of the membrana tympani, which become greatly distended, and the accompanying stretching of the tense and sensitive tissue in which this occurs accounts for the pain.

Fig. 135 represents the anatomical nervous connection between the teeth and the membrana tympani.

It is thus seen that a great many symptoms, usually of slight import, but marked enough to give much discomfort to the infant, may arise during this period of dentition, when the infant's entire nervous system seems to be in a very sensitive condition.

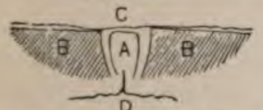
Gum-Lancing.—The question of lancing the gums during the period of dentition is one which has given rise to much discussion and to very diverse opinions. In former times it was erroneously believed that the teeth played an important part in almost every disease which occurred in early life. It was also supposed that lancing the gum relieved the symptoms of these diseases in some unexplained way. This extreme view soon had to be modified, and of late years many observers have come to the conclusion that it is never necessary to lance the gums. In cases of difficult dentition, however, as just explained, irritation arises very commonly in the later stages of the development of a tooth, and the question therefore remains whether this irritation in various parts of the economy, notably in the ear, can be relieved by lancing the gum. With regard to the question of gum-lancing, it may be said that it should be resorted to only under very exceptional circumstances.

During the dental period two classes of irritation are met with in connection with the teeth: (1) *irritation of the dental nerves*, with symptoms of reflex aural disturbance; and (2) *irritation of the gum* over the crown of the tooth from pressure, with symptoms of local irritation. We here have two entirely different conditions. If, when pain or symptoms in some other part of the economy seem to arise from dental irritation, we find that the gum which covers the crown of the still undeveloped tooth is soft and flat as in other parts of the mouth where a tooth is not about to come through, lancing the gums is manifestly absurd, as there is evidently no reason for making a wound in the mouth.

The second class of cases, however, although rather rare, must still be recognized as distinct in themselves and requiring especial treatment. In this class it is very evident that the gum for some reason does not give way to the growth of the tooth. Where the gum covers the crown of the tooth the tissues are swollen, tense, almost cartilaginous in their feeling, and hot. When this combination of abnormal conditions is found over the crown of the tooth, it can be relieved at once by the lancet.

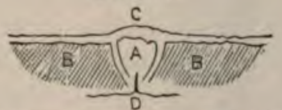
Figs. 136 and 137 represent the condition of the gums in relation to the teeth in the two classes of cases which have just been mentioned. Fig. 136 represents the mucous membrane over the crown of the tooth as flat and on a level with the rest of the gum. This is the condition of the gum in the majority of cases of difficult dentition, yet very severe symptoms of disturbance of the ear and cerebral circulation may apparently arise in these cases. The symptoms, of course, are very varied, the most definite ones being connected with the ear. In this class of cases the gum should never be lanced, even for the purpose of bleeding; as the mouth is not a fit place for such a procedure. The treatment of these cases should be directed to the especial part of the economy from which the symptoms arise. For instance, if the ear is affected, the indication is to relieve the reflex congestion. This can be done by the instillation into the ear of a few drops of an atropine solution, such as in the following prescription:

FIG. 136.



A, tooth in bone socket; B, jaw-bone; C, gum, soft, not inflamed or swollen; D, dental nerve.

FIG. 137.



A, tooth in bone socket; B, jaw-bone; C, gum, tense, inflamed, swollen; D, dental nerve.

PRESCRIPTION 83.

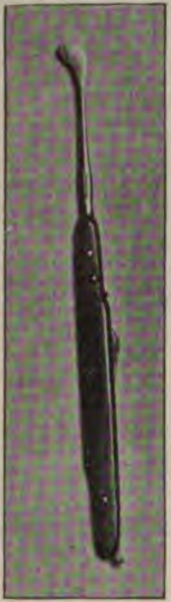
Metric.	Gramma.	*Apothecary.*	
℞ Atropinæ sulphat.	0.06	℞ Atropinæ sulphat.	gr. i;
Glycerini,		Glycerini,	
Aq. destil.	āā 3.75	Aq. destil.	āā ℥i.
M.		M.	

Sig.—Drops for aural congestion.

In addition to this, bromide of potassium should be given in repeated doses to the extent that is indicated by the especial case.

In Fig. 137 the mucous membrane covering the crown of the tooth is shown to be markedly raised above the level of the gum. In these cases, symptoms of local origin and often of great severity arise. The infant evidently has extreme pain and tenderness in its mouth. It cries incessantly, and often refuses to take its nourishment, on account of the acute pain which it suffers, and also of the tenderness which is produced by the least pressure on the gum, so that it may become weak and exhausted. There is usually a considerable elevation of the temperature, to 38.8° C. and even 39.4° and 40° C. (102°, 103°, and 104° F.). Vomiting is not uncommon, and there is twitching to such an extent that convulsions seem to be threatening, and at times actually occur. There are also great restlessness and insomnia.

FIG. 138.



Gum-lancet.

In these cases lancing the gum affords immediate relief. The temperature quickly subsides, the pain and general nervous symptoms disappear, and the infant after sleeping quietly for an hour or so wakes up and takes its food with avidity. The treatment in this class of cases, when the diagnosis is once made, is to lance the gum. This is done in the following way. The infant is placed in the nurse's lap, with its head in the lap of the physician, the nurse holding its arms firmly. The physician, after having first thoroughly sterilized his hands and washed the infant's mouth and gums with sterilized water, carefully makes an incision over the swollen gum well down to the crown of the tooth. Fig. 138 represents the gum-lancet which I am in the habit of using for this purpose. As only the end of this lancet is sharp, there is less danger of wounding the infant's lips and mouth than with the ordinary bistoury. Before using the lancet it should be thoroughly sterilized.

Although much has been said about the danger of hemorrhage in these cases, and of infection of the wound by pathogenic organisms, yet instances in which such results have occurred are so exceedingly rare that they should not deter us from treating the case properly as we would treat an abscess in the mouth, tonsil, or pharynx. It has also been said that a cicatrix may form on the gum over the crown of the tooth as a result of lancing. This is an exceedingly rare occurrence, and need scarcely be taken into account. The probability is that, when such an instance has occurred, the case was not one in which the gum should have been lanced, and the fear of such a result as this should certainly not weigh in the balance against the possible exhaustion and acute pain which may continue for days unless relief is given by cutting.

It must be understood, however, that the first class of cases of difficult dentition are by far the most frequent.

The following case is illustrative:

An infant, ten months old, was brought to the clinic with the following history:

It had one lower incisor. At the time when this tooth was about to appear above the margin of the gum the infant was very restless, and had considerable fever, and pain in its ear. Somewhat later a muco-purulent discharge came from the ear, but the general symptoms of restlessness, pain at times, and the local symptoms of heat and irritation in the mouth continued until just before the tooth had pierced the gum. After that time the discharge from the ear ceased, and the infant became perfectly well, the local irritation also having disappeared.

Three or four days previous to being seen at the clinic the same symptoms returned. The infant was evidently suffering from irritation in its mouth. Sometimes the gums were hot and dry, and again there was a hypersecretion of saliva. It continually put its finger to the gum of the lower jaw, sometimes almost locating it near the place where the first tooth was cut. The ear had begun to discharge again, and the infant showed signs of general discomfort by rubbing its nose and head continually and at times crying out as though in pain.

On examining the gum it was found to be swollen, but there was no especially tender point. On examining the ears an old perforation of the membrana tympani was found in the right ear, which was discharging, while in the left ear there was a simple congestion.

Such cases as this are often treated by lancing the gum, yet this procedure is not of the slightest use, and is, in fact, contraindicated, as it will only increase the already existing irritation of the mouth. The treatment should be the internal administration of bromide of potassium and appropriate local treatment for the ear.

The other cases are so similar and are so commonly met with that I need not dwell upon them, but shall report one of the cases in which lancing of the gum is indicated.

An infant, eight months old, and in good health, cut its first tooth when it was seven months old. At this time there were no nervous disturbances, the tooth coming through the gum without any reflex or local symptoms whatever.

When the second tooth was pressing on the gum I was called to relieve the following symptoms. The infant, who had been perfectly well, and who on examination showed no disease of any organ, was reported to have been feverish, restless, and crying out with pain for the previous twenty-four hours. It had refused to nurse, had not slept for thirty-six hours, had vomited a number of times, and was found to have a temperature of 40° C. (104° F.). It twitched from time to time, and apparently was in danger of having general convulsions. On examining the mouth one of the lower middle incisors was found to be entirely through the gum. The gum next to this incisor was greatly swollen, tense, cartilaginous in feeling, hot, and tender, so that whenever it was touched the infant screamed with pain. I then lanced the gum. The expression of pain, which had been marked on the infant's face, disappeared immediately, and was replaced by an expression of tranquillity, and it was evident that the severe pain had been relieved instantaneously. The infant went to sleep at once, and slept two hours. When it awoke its temperature was normal, it took the breast with great eagerness, and from that time it had no more trouble in its mouth. All the rest of its teeth were cut without any abnormal symptoms.

The following case illustrates to a still greater extent the necessity of lancing the gums in certain cases.

An infant began to have irritation from its teeth when it was five months old. At this time it woke up in the night screaming, and continued to scream with pain for several hours, during which time its parents had to walk continually up and down the room with it. Various remedies were administered, but without the slightest relief, and finally, after two days of suffering, in which it refused to take its nourishment, it lost in weight, and seemed very ill. An incision was made over the hot and swollen gum, with immediate relief.

The same symptoms occurred when the next tooth appeared beneath the surface of the gum, but were relieved, after waiting for a few hours, by lancing. Of the remaining eighteen teeth, six or eight gave rise to similar symptoms, but in every instance immediate relief was afforded by the lancing of the gum.

DISEASES OF THE NOSE.

The nose is the normal passage for the entrance of air to the lungs, and it is principally here that the air is modified before entering them. In normal respiration the mucous membrane of the nasal cavities, on account of the peculiar shape of the turbinated bones, presents a large surface to the inspired air, and is therefore admirably adapted to filter it of particles of dust and micro-organisms. The air is also warmed and changed so that before it reaches the larynx it is saturated with moisture and heated to a temperature of 35° C. (95° F.). This modification of the air is especially important in the new-born, since the lung has so lately been brought into use and is in such a comparatively undeveloped condition that it cannot withstand unchanged air, to which it adapts itself better later in life. The passage through which the air passes in going to and through the naso-pharynx is extremely narrow in young infants, and can easily become occluded. There are not many diseases which occur in the nose in infants and young children, and those which we do find are serious chiefly by being the cause of occlusion. In case of mouth-breathing due to nasal occlusion in an infant, the air which has not been modified by passing through the nose and naso-pharynx may have a detrimental influence on the lung and general circulation, thus striking a serious blow at the infant's vitality. In later childhood, although the occlusion which arises in the nares may not be so serious as regards the life of the patient, yet the results of such a condition will be represented by retarded development of the child and interference with the function of hearing, with resulting mental dulness.

RHINITIS.

The most common pathological condition which occurs in the nose in infancy and childhood is some form of rhinitis. This may be acute or chronic, catarrhal or purulent, hypertrophic or atrophic. New growths are rare. Of these the more common is myxoma or simple mucous polypus.

Acute Rhinitis.—Acute rhinitis (acute coryza) is an inflammation of the mucous membrane of the nasal cavities.

Etiology.—The cause of the disease in most cases is apparently undue exposure to cold, though it may be proved eventually that this exposure merely prepares the way for the attack of some micro-organism. The frequency with which a cold in the head follows a similar condition in an attendant or a companion, suggests strongly a direct infection. This condition may in almost all cases be considered as part of a disease which affects the mucous membrane of the naso-pharynx as well as of the nares.

SYMPTOMS.—The symptoms are a sense of fulness, burning, and dryness in the nostrils, succeeded in a few hours by a serous discharge, which later becomes muco-purulent. There is usually a slight rise of temperature, and, although the general symptoms are often slight, there is commonly a very evident sense of discomfort, with loss of appetite and general malaise. In some cases, by direct extension of the inflammation through the Eustachian tube, an otitis media may be caused. The entrance of air into the naso-pharynx is blocked by the swelling of the erectile tissues covering the turbinate bones, and almost complete occlusion takes place. The patient is then forced to breathe with the mouth open, and a resulting condition of dryness of the mucous membrane of the mouth and throat and a choking sensation arising from it follow. The natural tendency of an infant or young child is to keep the mouth shut, so that often when the nose is occluded it breathes with great difficulty when asleep, and its face becomes congested and even cyanotic. On forcing the mouth open the symptoms of congestion and cyanosis disappear, and the child begins to snore, and breathes with comparative comfort so long as its mouth remains open, until the dryness of the throat wakes it up.

PROGNOSIS.—The prognosis in these cases of acute rhinitis is usually good. The disease runs its course in a variable period of from three days to a week, and, unless the child is subjected to fresh exposure, it recovers entirely. The prognosis, however, varies in accordance with the age of the individual attacked. The danger that a young debilitated infant may die from exhaustion when the nares are occluded is considerable, and I have seen a puny, ill-cared-for infant die of a simple acute rhinitis with occlusion. Instances of this kind should warn us that active treatment is indicated.

TREATMENT.—The treatment should be directed primarily to relieving the nasal occlusion. This is best accomplished by atomizing the nose. In most cases the oil atomizer containing oleum petrolatum album is sufficient to afford relief. In addition to the local treatment, the administration of stimulants is indicated when there is exhaustion. Care should be taken that the infant is taking a sufficient amount of nourishment. This is especially difficult to determine if it is nursing, as under these circumstances it will often hold the nipple in its mouth and apparently suck, while its breathing is so much disturbed by the nasal obstruction that it does not draw much milk from the breast. The various drugs which have been recommended for acute rhinitis have not in my hands proved to be of much use. I have occasionally found that a few drops of the tincture of euphrasia repeated three or four times at intervals of an hour will seemingly lessen the nasal secretion.

An instance of this class is the case of an infant who had an attack of acute rhinitis when she was four months old. Although she was well nourished and fairly to the other. As would naturally be expected from the lesions, the symptoms are those of restlessness, especially at night, and various reflex phenomena connected with the throat and the larynx. Thus, there may be continued cough, and, when the Eustachian tubes are occluded, deafness and a resulting hebetude. At times interference with speech results. There is not much nasal secretion in these cases, which aids us in the differential diagnosis from the other forms of rhinitis which have just been mentioned.

TREATMENT.—The treatment of these cases when they are dependent upon growths in the naso-pharynx is the surgical removal of such growths. Mild astringent sprays should be used, and the oleum petrolatum spray recommended in catarrhal rhinitis. As a rule, these cases should be placed in the hands of a specialist.

Atrophic Rhinitis (*ozæna*).—By atrophic rhinitis is meant a condition of the nose characterized by atrophy of the mucous membrane and of the bony prominences within the nose, accompanied by what has been termed a dry catarrh, as a result of which the secretion of the nose forms crusts, which undergo decomposition and become fetid. It is also called ozæna.

ETIOLOGY.—The disease is one which attacks older children rather than infants, and its etiology is obscure. The glandular function is impaired, and the muco-purulent discharge becomes thick and firmly adherent in the form of crusts to the sinuosities of the nose. This film of desiccated muco-pus in drying contracts the underlying turbinated tissues in such a way as to interfere with the circulation of the blood, a condition which limits glandular action still more and conduces to general atrophy.

SYMPTOMS.—The symptoms of atrophic rhinitis are the formation of crusts and the presence of fetor.

TREATMENT.—Although the tissues which have actually been destroyed by the atrophic process cannot be restored by treatment, the patient can be entirely relieved of the crust formation and fetor by persistent and patient local washing and applications. The details of treatment differ according to the extent and character of the disease. Crusts may be removed by spraying or douching, great care being taken to prevent the washing fluid from entering the Eustachian tubes. If this is not sufficient to remove the crusts, the nasal cavities must be illuminated with a head-mirror, and the crusts carefully brushed off with a cotton-stick. The formation of dry, hard crusts is often prevented by frequent spraying with an oil. Local applications of different substances are of use in many cases, but these should, as a rule, be carried out under the direction of a specialist in the treatment of diseases of the nose.

MUCOUS POLYPUS.

This is a pedunculated connective-tissue growth originating from the mucous membrane of the middle turbinate bone. It is rare in children, It does not grow on a healthy mucous membrane, and is always preceded by some morbid condition of the nose. It is often multiple.

The symptoms begin with a nasal discharge followed by nasal occlusion. The diagnosis is easily made by a mirror and a probe. The treatment is the removal of the growth.

EPISTAXIS.

During the period of early childhood epistaxis, or hemorrhage from the nose, is not uncommon. I have occasionally met with epistaxis in young infants, but in my experience it is rare in the early months of life. In older children recurrent epistaxis, especially if unilateral, points to the presence of an erosion or a varicose condition in the cartilaginous septum near the external opening of the nose.

Unless the individual happens to be affected by hæmophilia, epistaxis is not especially dangerous, and usually its occurrence ceases as the child grows older.

TREATMENT.—The application of pressure on the side of the base of the nose and the use of ice are usually sufficient to stop the hemorrhage. If the epistaxis is due to the varicose condition just spoken of, it can be readily controlled temporarily by a plug of cotton pressed upon the bleeding part. For a permanent cure, cauterizing the bleeding part may be necessary.

DISEASES OF THE EAR.

It is very important to bear in mind, in examining infants and children, the common occurrence of some morbid process in the ear. In many cases in which the more pronounced aural symptoms are not evident, symptoms which appear obscure, but really are due to some latent disturbance in the neighborhood of the ear, reflex or otherwise, are readily explained when in addition to the presence of some other disease the unusual symptoms are found to arise from the aural complication. The question of diseases of the ear in infancy and childhood has not received from the general practitioner, nor, indeed, from those who devote themselves especially to children, the attention that it deserves. Even leaving out of consideration the cases of disease of the middle ear incident to the exanthemata, serious implications of the ear from other causes are not uncommon during the first year of life, and we should especially watch for an aural complication in pneumonia.

Von Tröltsch found on examining forty-seven petrous bones taken from twenty-four unselected children that the middle ear was normal in only eighteen. The other twenty-nine ears showed in varying degrees the appearance of a purulent and sometimes, though rarely, of a mucous catarrh. Of the fifteen children with exudation in the middle ear, the youngest was three days and the oldest one year old: five were in their first month, two each in their second and fourth, three in their third, and one each in their seventh, eighth, and twelfth months.

In every five examinations of the ears of new-born children Schwartze found the tympanum filled with pus in two.

Wreden found in eighty ears of children a normal middle ear in only fourteen; purulent catarrh existed in thirty-six, and simple mucous catarrh in thirty; the youngest child had lived twelve hours, the oldest fourteen months. The majority of these cases were, however, from three to fourteen days old.

Edward Hoffman examined twenty-four petrous bones in infants varying in age from thirty-two hours to four weeks, and found the tympanum filled with pus in seven cases.

Of two hundred and thirty carefully examined cases under seven months of age Kutcharianz found the tympanic mucous membrane normal in thirty only. In fifty it showed either slight or intense catarrhal inflammation, and in one hundred and fifty the tympana were filled with pus.

These statistics, quoted from Von Tröltsch, serve to emphasize the statements of that author that even from the beginning of extra-uterine life "there is an unusually strong disposition to disease of the middle ear, owing on the one hand to the double influence of the peculiar morphological relations of the ear and the pharynx, and on the other hand to the diseases and conditions of life to which the child is frequently exposed."

These pathological and clinical observations emphasize the fact that the ear in children is a very frequent source of infection by a number of pathogenic organisms, such as the pneumococcus and streptococcus, and that this infection, although frequently primary, is usually secondary to infection elsewhere, as from the cerebral meninges and especially from the naso-pharynx.

We should therefore consider carefully the ear in all cases in which the symptoms are obscure, as well as in those diseases in which it is well known that aural complications are liable to arise.

DISEASES OF THE NASO-PHARYNX.

Although the cavity of the naso-pharynx is small and apparently insignificant, yet it plays a very important part in a number of the diseases to which children are susceptible. The condition which makes this portion of the respiratory tract especially important is the presence of the pharyngeal tonsil which lines its cavity.

HYPERTROPHY OF THE PHARYNGEAL TONSIL (Adenoid Growths).

The glandular or lymph-tissue which lines the vault and posterior wall of the naso-pharynx is very similar to that which composes the faucial tonsils, and is called the pharyngeal, third, or Luschka's tonsil. Under certain circumstances this tissue becomes hypertrophied, and gives rise to the condition which is usually designated as adenoid growths.

Etiology.—The pharyngeal tonsils, like the faucial tonsils, are normal structures, and become pathological only when they are hypertrophied or diseased. Careful examination will reveal the presence of hypertrophy in the majority of children, but in normal cases it is small and produces no symptoms. Although hypertrophy of the pharyngeal tonsil may occur in infancy, it is uncommon before the second or third year. The disease is essentially one of childhood, as it very seldom develops after puberty. Acute inflammatory conditions or some obstruction in the nose are probably the inciting causes of the hypertrophy.

Pathology.—The pathological condition which is found in the lymph-tissues of the naso-pharynx is an hypertrophy which is very similar to the hypertrophic condition of the faucial tonsils, except that the latter contains a greater amount of connective fibrous tissue. The hypertrophy may be of greater or less extent, sometimes not being sufficient to cause any especial occlusion and at other times completely occluding the posterior nares.

Symptoms.—The most prominent symptom noticed in children who have this disease is that they breathe with their mouths open at night and snore. As the nares become more occluded the child begins to breathe through its mouth also when it is awake. The interference with the proper passage of the air to the larynx and lung results in a chronic form of pharyngitis and laryngitis, while the blocking of the nasal end of the Eustachian tubes may result in a chronic catarrhal condition of the middle ear. Any or all of these symptoms may arise in an individual case according to the amount or position of the obstruction. The child's expression changes, and is almost characteristic when the disease is fully developed. It holds its mouth open, and the lower jaw appears to drop, giving it a stupid look. If this condition continues after the seventh or eighth year, the bridge of the nose is apt to be prominent and its sides to look pinched; the palate may be markedly arched, and the upper jaw narrowed laterally so as to crowd the teeth. The faucial tonsils may or may not be enlarged, but are usually so. Another symptom is an increased liability to attacks of acute rhinitis, and a tendency for these attacks to become subacute or chronic.

Lack of development of the chest with flattening of the front of the thorax may be caused, not, as was formerly supposed, by the enlargement of the faucial tonsils, but by the occlusion caused by the hypertrophy of the pharyngeal tonsil. This hypertrophy with its resulting nasal occlusion may also be the cause of pharyngitis, laryngitis, and perhaps of bronchial catarrh or asthma, which can be cured only by the removal of the primary cause, the pharyngeal tonsil.

Diagnosis.—The diagnosis of hypertrophy of the pharyngeal tonsil is not difficult in a marked case or if it is possible to examine the child's naso-pharynx. In young infants the posterior nasal space is so minute that it is almost impossible to reach it. The diagnosis can often be made simply by the appearance of the child, as there is no other disease which especially simulates this condition. A definite diagnosis, however, can be made only after the hypertrophied tonsil has actually been seen or felt.

The importance of learning to detect by means of the finger the presence of an enlarged pharyngeal tonsil is great. If an examination with the posterior rhinoscopic mirror can be made the exact extent of the hypertrophy can be seen, and the child spared the discomfort of a digital examination, but the examination with the mirror in the throat is usually so difficult in young children that the direct detection by means of the finger is often the best method. The child should have a blanket pinned around it tightly, so as to keep it from moving its arms. It should be held firmly sitting in the nurse's lap. The child's head is then held with one arm, pressing the cheek between the back teeth with the forefinger, then passing the forefinger of the other hand gently, firmly, and quickly over the base of the tongue and behind the soft palate until it reaches the posterior wall of the pharynx. By quickly turning the finger upward, it is easy to feel whether the cavity of the naso-pharynx is clear or whether it is more or less filled by a soft, spongy mass, the hypertrophied pharyngeal tonsil. There is usually a little blood on the finger when it is withdrawn, as the growth is friable and bleeds easily. This examination is not, as a rule, very painful to the child, but is in many cases very disagreeable and alarming to it. When the finger is once in the mouth, it is not wise to take it out again until the examination is completed, as the child can rarely be induced to allow a second attempt to be made. In passing the finger over the base of the tongue it is necessary to be careful to get the finger behind the soft palate, and not to push it upward and backward, for in this case the soft tissues of the palate may feel like an adenoid growth. The child can be prevented from biting the finger by simply keeping the cheek pressed between the teeth as has just been described.

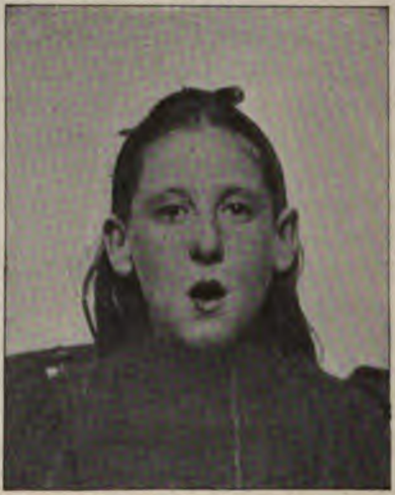
Fig. 139, page 648, represents the typical picture of an adenoid growth in a girl ten years old. She held her mouth open and evidently had complete occlusion of the posterior nares, the anterior nares on examination being found entirely free. The pinched look of the face on either side of the nose and the prominence of the bridge of the nose are to be noticed. The child was dull, the dulness having increased as the other symptoms of the adenoid growth developed, and her face had a stupid expression. On examining the child's mouth the hard palate was found to be very much arched, the tonsils were enlarged, and the soft palate was slightly pushed forward, the pharynx being narrower than normal.

After removal of the adenoid growth and faucial tonsils she found no difficulty in keeping the mouth closed, not only while awake, but also at night, and slept much more quietly than before. Her general health improved, and the development of her face during the remainder of its growth was normal.

Prognosis.—The prognosis of cases of hypertrophy of the pharyngeal tonsil varies greatly, for there are all forms and degrees of the affection.

In some cases the swelling of the lymph-tissues occurs only at intervals when the child has been subjected to exposure in inclement weather; it will then show itself simply by occlusion of the nares, with resulting discomfort, lasting for some weeks, but disappearing eventually as the weather becomes milder or if the child is taken to a different climate. The naso-pharynx has an important function besides being a passage-way for the air. It lubricates the pharynx, and by the action of its muscles opens the Eustachian tubes during the acts of swallowing and yawning, thus ventilating the ear. The prognosis, therefore, must vary according to the degree in which any of these functions are interfered with. When infants, before they have learned to articulate, become deaf they may gradually lose the power of speech, and from being deaf they may fall into a condition of hebetude which sometimes closely simulates idiocy, though it is not true idiocy, for the mental condition quickly changes when the cause of the disturbance has been removed. Unless irreparable injury has been done to the ear or to the general development, the prognosis is very favorable, provided the proper treatment is carried out.

FIG. 139.



Hypertrophy of pharyngeal tonsil (adenoid growths). Female, 10 years old.

TREATMENT.—The best treatment of these cases is to remove the adenoid growth at once. The operation in the hands of a skilful surgeon is not dangerous, and should be unhesitatingly advised. There are a number of methods which have been employed in operating on these cases. The child should be etherized, or, as is preferred by some operators, chloroform may be employed. Most operators prefer to have the child held sitting in the lap of an attendant, others to have it lying down with its head bent backward. Dr. French, of Brooklyn, uses an especial chair adapted by him for operating in the upright position. There is less liability to extreme hemorrhage and to infection of the middle ear through the Eustachian tube in the upright position, and the danger of inspiring the blood, which has been given as a reason for operating in the prone position, is now well known to be visionary. The instrument which is commonly used is the Gottstein curette, which acts on the principle of a draw-knife, held in a stirrup-shaped frame; or a pair of post-nasal forceps held in the right hand is introduced, closed, into the naso-pharyngeal cavity. The blades are then opened, and pieces of the mass are grasped one after the other and pulled off gently: under no circumstances is force to be exerted. With proper care and assistance there is no danger to the child, and often in a few minutes a morbid condition which has existed for years may be practically cured. There are, of course, many details in this operation which must be thoroughly understood in order that it should be successful. They need, however, scarcely be mentioned here, as the operation should be performed only by one whose work has especially fitted him for it.

These growths when not extensive are sometimes removed even without ether with the curette or the finger-nail.

The following case illustrates the benefit of operative treatment for the removal of the pharyngeal tonsil when hypertrophied.

When the child was first seen at two years of age he had a very bright expression, and spoke well; he showed nothing abnormal in connection with the shape of his nose or face. He heard well, slept with his mouth shut, and had a free passage of air through a perfectly normal nose and naso-pharynx. When he was three and a half years old, however, it was noticed that he snored at night, breathed with his mouth open, and was subject to continual attacks of rhinitis and naso-pharyngeal occlusion. Following these attacks his hearing became affected, and, while in his second year he had been bright and always ready to play with his parents, he became dull, and did not care to play with others, but would sit for hours playing by himself with his toys.

A digital examination showed a mass of considerable size blocking the posterior nares. On the removal of this mass, which proved to be an hypertrophied pharyngeal tonsil, rapid improvement took place in his general condition, the dulness and hebetude disappeared, and he returned to the normal condition which he represented in his second year.

There are other growths which occur in the naso-pharynx, such as sarcomata. They are, however, too rare to need especial description.

DISEASES OF THE PHARYNX.

Diseases of the pharynx in children are especially those affecting the tonsils, the uvula, the soft palate, and the posterior wall of the pharynx.

TONSILLITIS.

By tonsillitis is meant an inflammation of the tissues of the faucial tonsils. This inflammation may be acute or chronic.

Acute Tonsillitis.—Acute tonsillitis (*acute follicular tonsillitis, acute cryptic tonsillitis*) is characterized by an acute swelling and redness of the glands, and an exudation from the crypts. It is generally bilateral, but one tonsil may be affected before the other. The disease is not limited to the faucial tonsils, but commonly involves neighboring glandular structures, such as the pharyngeal and lingual tonsils.

Etiology.—The cause of this form of tonsillitis is undoubtedly infection by some pathogenic germ. It is probable that more than one form of germ is capable of causing it. Many of the pathogenic germs which infest the mouth or the throat may be found in the crypts in this disease, but the especial germ by which we can characterize the disease has not yet been determined.

Symptoms.—As a rule, the disease is characterized by an acute onset, with a heightened temperature, 39.4° to 40° C. (103° to 104° F.), loss of appetite, and general malaise. I have often noticed, however, that the symptoms of a marked follicular tonsillitis are not so acute and do not so definitely point to the throat in young children as they commonly do in older children and in adults. In fact, in many cases, unless the throat is actually inspected, it would seem as though it were not a local affection of the throat, but some general disease affecting other parts of the system. We should be especially careful, therefore, not to have our attention diverted from the throat, but under all circumstances, where these symptoms arise in young children, even though they apparently swallow without discomfort, the throat should be examined before deciding whether or not some other disease is developing.

On examining the throat the tonsils are seen to be enlarged, reddened, and in the early hours of the disease to show a little swelling of the orifices of the crypts, as though a secretion within them was about to burst the overlying mucous membrane and appear on the free surface. Later this actually occurs, and the tonsils are seen to be studded with white or grayish-white spots. These do not appear on the soft palate or uvula, though they may appear on the pharyngeal tonsil, the base of the tongue, and the posterior pharyngeal walls. The mucous membrane of the pillars of the palate, of the uvula, and of the soft palate are usually reddened, and there is very apt to be a decided reddening and even swelling of the mucous membrane and follicles of the pharynx. As the disease progresses these spots may coalesce and, adhering to the surface of the tonsil, form a pseudo-membrane which is often impossible, without a bacteriological examination, to distinguish from diphtheria. As there is a direct connection between the tonsils and the cervical glands, the latter are liable to be involved, though any great swelling of the cervical glands in connection with acute tonsillitis is uncommon.

The disease is self-limited, and runs its course in two or three days or a week, at the end of which time the general symptoms subside, the appetite returns, the temperature becomes normal, and the child, although it is left somewhat weakened by the disease, seems as well as ever. The tonsils themselves, however, may not for some time regain their original size, and the exudation often remains in the crypts and may cause a chronic irritation with a tendency to recurrence.

Diagnosis.—The differential diagnosis of follicular tonsillitis is to be made from the various forms of stomatitis, which have already been sufficiently described, and from diphtheria. It is now very generally known that it is impossible absolutely to exclude diphtheria by the morbid appearances seen on the tonsils. In the great majority of instances, however, when the attack is acute, when the cervical glands are not especially involved, when the white spots on the tonsils are clearly located in the orifices of the crypts, and when there is no appearance of a membrane on the soft palate or pharyngeal wall, we can make a provisional clinical diagnosis of follicular tonsillitis with considerable confidence, but never with certainty without a bacteriological examination.

Prognosis.—The prognosis of follicular tonsillitis is in almost every case favorable, and is rendered unfavorable only by the complication of a peritonsillar abscess, but it should be remembered that in an inflamed tonsil pathogenic organisms, such as those of diphtheria, are more apt to develop than when there is no inflammation.

Treatment.—The treatment of acute tonsillitis, according to my experience, should be entirely symptomatic. It is a self-limited disease, and in a vast majority of cases is not benefited by the administration of any drug internally or by local applications. In order to avoid the invasion of the various pathogenic germs during the progress of the tonsillitis I am in the habit of having the throat kept thoroughly clean with mild solutions of chlorate of potassium or borate of sodium. In young children, as a rule, I make no local application beyond allowing them to swallow cold solutions of chlorate of potassium in the strength which I have already advised (page 624). Small doses of quinine according to the age of the child are indicated when there is much exhaustion or malaise following the attack.

The local application of a cleansing spray, and the administration of ice if desired to relieve the discomfort, constitute all that is necessary for the treatment of most cases. It is best not to disturb the mucous membrane with applications on swabs or brushes.

The following case represents a follicular tonsillitis:

A child (Plate XI., facing page 620, Follicular Tonsillitis), four years old, had an attack of follicular tonsillitis.

She was taken sick two days before entering the hospital, with a heightened temperature of about 39.4° C. (103° F.), loss of appetite, and general malaise. She did not complain of her throat, and swallowed without difficulty. Nothing abnormal was found in any of the other organs, but on inspecting the throat the tonsils were seen to be enlarged and much reddened, and one or two of the orifices of the crypts were somewhat raised above the general surface of the tonsil. On the following day a number of white spots of different sizes appeared on both tonsils. On the day of entrance the redness was mostly confined to the tonsils, and affected the uvula and palate very little. On the inner surface of both tonsils the exudation had coalesced.

so that it had an appearance very much like that of a pseudo-membrane. It is not uncommon in follicular tonsillitis for this coalescence of the exudation to take place on the surface of the tonsil which points towards the median line of the throat. The other appearances of the tonsils were characteristic of follicular tonsillitis. On the upper left-hand corner of the left tonsil, close to the arch of the palate, was an enlarged cryptic orifice which had not quite broken down, and which appeared as a light red prominence on the general surface of the tonsils. The orifices had a like appearance in various parts of both tonsils. On the anterior surface of the left tonsil were two white spots, caused by the exudation from the crypts. In the upper part of the right tonsil were three smaller yellowish-white spots, and lower down on the tonsil a grayish-white rather large spot, all of them due to the same cause. There were no other lesions in the throat, and the cervical glands were not involved.

Cultures made from this exudation did not show the presence of the Klebs-Loeffler bacillus.

The other affections of the tonsils, such as occur in the course of the exanthemata and in diphtheria, constituting the pseudo-membranous form of the disease, can best be described in connection with the especial diseases in which they arise.

Chronic Tonsillitis.—The terms chronic tonsillitis and hypertrophied or enlarged tonsils are commonly used to express the same condition, especially in children, in whom a chronic inflammation of the tonsils unaccompanied by enlargement seldom occurs.

Although this hypertrophy of the faucial tonsils may exist without a corresponding affection of the pharyngeal tonsil, yet it is very apt to be associated with this latter condition.

PATHOLOGY.—The enlargement is a true hypertrophy, sometimes accompanied by more or less inflammatory deposit. If the parenchymatous or glandular tissues are especially affected we find a soft, more or less red and vascular tonsil, with large crypts, often containing much secretion. In the interstitial form the tonsil is hard and tough, the crypts less prominent or even very small, and the vascularity much diminished. These types are the two extremes, and are rare; in most cases the enlargement is essentially one of hypertrophy. The tonsils may be only moderately enlarged, or their size may be so increased that they meet, touching each other in the median line.

SYMPTOMS.—The symptoms of hypertrophy of the faucial tonsils vary according to the degree of enlargement. Normally the tonsils can scarcely be seen on inspection of the throat. In examining a child for enlargement of the tonsils care must be taken that the pharynx is not contracted by gagging at the time the examination is made. The act of gagging, which is easily brought about in children by a careless use of the tongue-depressor, brings the tonsils towards the median line, thus giving tonsils of normal size the appearance of being large and obstructive. When only moderately enlarged they may produce no symptoms whatever, or they may be irritated by various external influences and be the source of recurrent acute affections of the throat. When considerably enlarged they may still not produce any marked symptoms, provided that the passage of air through the naso-pharynx is unobstructed. They may, however, even when the pharyngeal tonsil is not enlarged, cause obstruction in the naso-pharynx by pressure as they enlarge upward and backward. When this happens, the same interference with the breathing and development of the child takes place as when the obstruction is primarily in the naso-pharynx. These symptoms are the same as have already been described in speaking of hypertrophy of the pharyngeal tonsil, and therefore need not be mentioned in detail here.

Occasionally difficulty in swallowing and thickness of speech may arise when only the faucial tonsils are enlarged.

PROGNOSIS.—The prognosis in cases of hypertrophy of the faucial tonsils depends upon these varied anatomical conditions just described. So long as the tonsils do not encroach on the naso-pharynx the prognosis, so far as injury to the child is concerned, is good. We should remember, however, that the enlargement of the tonsils is a fertile source of irritation which may prepare the way for serious disease produced by the various micro-organisms. The prognosis as to their disappearance is not especially good, as when once hypertrophied they seldom recover their normal size without active treatment, though they generally diminish slowly in size after puberty.

TREATMENT.—Local applications for the reduction of hypertrophied tonsils are useless. Some success has been obtained by Gampert by what is called discission of the tonsils. Leland has strongly advocated this treatment in certain cases. The operation consists in making slits in the tonsil with a knife especially devised by him.

The most thorough and certain way of curing the disease is, however, by excision. If the pharyngeal tonsil also is hypertrophied the child should have an anæsthetic, and the operation for adenoid vegetations and tonsils should be done at the same time. If the tonsils alone are enlarged, they may be excised with the tonsillotome without an anæsthetic, provided they are not adherent to the pillars of the fauces. After the operation the child should be made to gargle with a solution of borate of sodium for two or three days (Prescription 84), and should be given only milk for its food.

PRESCRIPTION 84.

Metric.	Gramma.		*Apothecary.*	
℞ Sodii Borat.	2	0	℞ Sodii Borat.	gr. xxx;
Glycerinæ	20	0	Glycerinæ	ʒ v;
Aq. destil.	180	0	Aq. destil.	℥ vi.
M.			M.	

Sig.—Gargle for throat.

The hypertrophied tonsils may have become adherent to the anterior or the posterior pillars of the fauces to such an extent that the guillotine either cannot be used, or not without danger of wounding these pillars.

In such cases it may be necessary to resort to dissection or to the galvano-cautery to remove the tonsillar tissue.

PERITONSILLAR ABSCESS.

Symptoms.—In some cases an inflammatory process resulting in suppuration occurs in the cellular tissue around, above, or behind the tonsil, constituting an abscess which is called *peritonsillar*. It is rather rare in early childhood. The disease is usually preceded by a certain degree of simple tonsillitis, and when it develops the temperature rises, perhaps to 40° or 40.5° C. (104° or 105° F.), and the child evidently suffers much pain, and has difficulty in swallowing or even in opening the mouth.

On inspecting the throat in these cases a unilateral swelling is seen in the neighborhood of the tonsil pushing the soft palate forward, and the tonsil towards the median line.

Prognosis.—The prognosis is in almost every case favorable, except in those which have been neglected or improperly treated. There is rarely extensive burrowing of the pus.

Treatment.—The treatment is to locate the abscess by careful palpation and to open it under strict antiseptic precautions with a guarded bistoury.

PHARYNGITIS.

An inflammatory condition of the posterior wall of the pharynx is rather rare in infancy, but is not uncommon in children. It is usually coincident with an inflammatory condition of the naso-pharynx or of the tonsils, but in a certain number of cases it is so much more pronounced in the posterior wall than elsewhere that it can be described as a separate disease. The diseases of the posterior wall of the pharynx are either (1) a simple catarrhal condition of the mucous membrane or (2) an inflammatory process in which the follicles are especially affected. In addition to these conditions, pus may form behind the mucous membrane, producing a retropharyngeal abscess. The inflammatory lesions of the pharynx may be acute or chronic.

The conditions which give rise to pharyngitis are the same various morbid processes that involve the parts in the neighborhood of the pharynx, such as the naso-pharynx and the tonsils. These causes have already been mentioned. There also seems to be a connection between certain irritations arising in the gastro-enteric tract and the pharynx. When this occurs it is usually the chronic form of pharyngitis which is met with, and the causal connection between these two distant parts of the economy is probably of a reflex nature.

Acute Simple Pharyngitis.—The pathological condition which is found in the simple acute form of pharyngitis is an acute inflammation characterized by a slightly heightened temperature, a hyperæmic condition of the blood-vessels of the posterior wall of the pharynx, and a certain amount of swelling and of serous exudation.

Symptoms.—The symptoms are discomfort in swallowing, and at first a feeling of dryness in the throat, followed later by an exudation of viscid mucus. The child does not seem especially sick with this disease, and the length of the attack varies according to the influences which are causing it.

Acute Follicular Pharyngitis.—Symptoms.—The acute follicular form of pharyngitis does not differ materially in its symptoms from the simple form, and in fact both forms are so frequently combined that a clinical distinction need scarcely be made between them. On examining the pharynx in this form of pharyngitis, in addition to the appearances which are seen in the simple form, the follicles will be found enlarged and raised above the surface of the mucous membrane.

Treatment.—The treatment of both forms of pharyngitis is essentially local, and is, as a rule, by applications to the inflamed mucous membrane in the form of a spray, either directly or indirectly through the nose. The spray is essentially for the purpose of cleansing and thus soothing the inflamed mucous membrane, and should consist of mild alkaline solutions such as the following:

Prescription 85.

Metric.	Gramma.	*Apothecary.*	
℞ Sodii chloridi.	0|3	℞ Sodii chloridi	gr. v
Sodii bicarb.,		Sodii bicarb.,	
Sodii boratis āā	0|9	Sodii boratis āā	gr. xv;
Aquæ rosæ	30|0	Aquæ rosæ	℥ i;
Aquæ	90|0	Aquæ	℥ iii.
M.		M.	

Sig.—Spray for pharyngitis.

Chronic Pharyngitis.—The chronic form of pharyngitis is usually accompanied by an irritating cough, which is most pronounced at night and in the morning. The mistake should not be made of thinking that these children who are coughing continuously, often losing in weight and looking weak and anæmic, are necessarily affected with bronchitis. This series of symptoms is frequently looked upon as diagnostic of bronchitis, when it really arises from pharyngitis, and can be cured by treating the latter disease.

Treatment.—The treatment of chronic pharyngitis is to remove any hypertrophic condition of the tonsils, tongue, or nose, to regulate carefully the child's general nutrition, and to avoid undue exposure to inclement weather or to air vitiated in any way, as by dust. Local applications of a one per cent. solution of nitrate of silver, followed immediately by thorough cleansing with sterilized water, are sometimes indicated in the more intractable cases.

Elongation of the Uvula.—Symptoms.—Accompanying pharyngitis, usually in its chronic form, an elongation of the uvula is at times met with in children. This condition may arise from a relaxed condition of the muscles of the soft palate and of the uvula, or may consist simply of a redundancy of the mucous membrane at the tip of the uvula. The general irritated condition of the uvula and the tickling sensation produced by its elongated tip touching the base of the tongue cause a harassing cough, which by its persistence weakens the child, gives rise to loss of appetite, and interferes with its nutrition.

A papillomatous growth is sometimes found attached to the tip of the uvula or to its side, which causes the same symptoms as elongation of the uvula. The treatment is excision, after which it does not recur.

TREATMENT.—Local applications of astringents, such as alum, are at times sufficient to restore the uvula to its normal condition, but the disease can be cured quickly by excising the end of the uvula with blunt-pointed scissors. The amputation of the entire uvula is to be avoided, as it has been known to weaken the muscular action of the soft palate.

RETROPHARYNGEAL ABSCESS.

Retropharyngeal abscess is a disease which occurs usually during the first year of life and is very rare after this time. The disease may be secondary to injuries of the pharynx, to abscess in the neck, and to disease of the cervical vertebræ, or it may be metastatic from septic processes such as occur in diphtheria. In a certain number of cases it is idiopathic so far as we know.

PATHOLOGY.—The pathology of the disease consists in the formation of an abscess in the tissues of the posterior wall of the pharynx, and is more apt to be on one side of the pharynx than in the median line.

SYMPTOMS.—The symptoms, whether the disease is primary or secondary, are very much the same. The first symptom is generally difficulty in swallowing, which may go on to entire inability to swallow. The infant is next noticed to breathe in a peculiar way. It holds its head back and its mouth open. The breathing may be described as snorting, and at times as almost stertorous, differing markedly from the whistling sound which is heard in obstruction of the larynx. On examining the throat the soft palate is seen to be pushed forward and to be somewhat anæmic. The posterior wall of the pharynx is bulging, usually unilaterally, is reddened, swollen, tense, and as the disease progresses is found to be fluctuating. In some cases the abscess burrows into the tissues of the neck and appears as a pear-shaped tumor behind the ear. I have met with two cases of this variety where the pus could be reached easily by an external incision.

DIAGNOSIS.—The diagnosis must be made chiefly from peritonsillar abscess. This is, as a rule, not difficult unless the latter condition has proceeded so far that the pus by burrowing has invaded the walls of the pharynx. It is usually not difficult to determine the situation of the abscess by passing the finger directly through the mouth to the posterior wall of the pharynx. If there is pus in the tissues of the pharynx a sense of fluctuation will be obtained. The position of the child in cases of retropharyngeal abscess is also significant, and is not that which is assumed in peritonsillar abscess. It holds its head back, in order to allow a free passage for the air through the occluded pharynx into the larynx.

The diagnosis must also be made from œdema of the glottis, but this is not, as a rule, difficult, for inspection shows that in the latter disease, bulging, redness, swelling, and fluctuation of the posterior wall of the pharynx are not present. The characteristic position of the head, also, is not seen in œdema of the larynx. A case of retropharyngeal abscess can often be diagnosticated by the snorting and labored character of the respiration.

Prognosis.—The prognosis in the cases in which the abscess is secondary varies according to the nature of the disease which causes it. Thus, it is an exceedingly dangerous complication in diphtheria, and is one of serious import in cervical spondylitis. In those cases of undetermined origin which are spoken of as idiopathic the prognosis is very good if the proper treatment is carried out at once. We must, however, consider the possibility of the abscess bursting suddenly and of the child suffocating by inspiration of pus into the larynx. This has been known to occur when the disease has been left untreated.

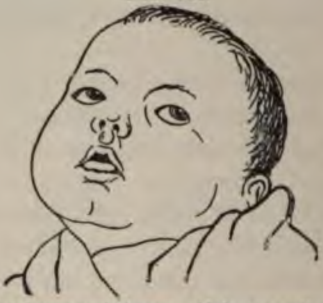
Treatment.—In the idiopathic cases the abscess should be opened at once. The method which I have found satisfactory in the cases which have come under my observation is to have an assistant hold the infant sitting upright in the lap, with a blanket tightly pinned around it so as to prevent it from moving its arms. Another assistant should hold the head. The mouth should then be opened, a guarded bistoury should be introduced into the pharynx and the abscess punctured. As soon as the opening has been made the bistoury should be removed quickly, and the infant's head should be immediately thrown forward and downward, so that the pus will be discharged from its mouth and not inspired into the larynx or swallowed. It is usually necessary after the operation to introduce the finger into the pharynx and to press the walls of the abscess, so as to empty any pus which may continue to collect there and also to keep the opening free. With this treatment, unless some complication should arise, the disease is usually cured in about a week.

Some operators prefer having the child placed in what is known as Rose's position, on its back with the head hanging over the end of the table.

Fig. 140 represents a typical case of retropharyngeal abscess in an infant seven months old, showing the characteristic appearance produced by the obstruction in the pharynx. The head was held back and the mouth open; the eyes were somewhat rolled upward, but she was perfectly conscious. The face was slightly cyanotic. On depressing the lower jaw and tongue the soft palate was seen pressed forward, and the blood-vessels were found to be almost empty and very pale. On the left of the median line a swollen, congested, bulging condition of the mucous membrane was seen on throwing a strong light from the mirror on the posterior wall of the pharynx. On touching the most prominent point of the swelling with the finger a sense of fluctuation was obtained.

The infant was said to have been sick for two weeks with a cold in its head. Three days before I saw it it began to breathe in a labored manner and to hold its head back rigidly and somewhat to the left. It also began to hold its mouth open. It was able to nurse only a few seconds at a time, when it would let go of the nipple and refuse to take it again. It had been growing very weak from lack of nourishment and from the exhaustion arising from the difficulty in breathing.

FIG. 140.



Retropharyngeal abscess. Male, 7 months old.

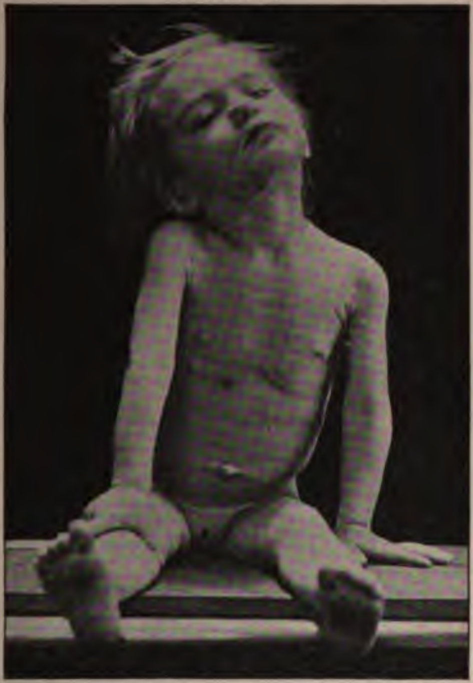
An opening in the abscess was made, and a large amount of pus was evacuated. An hour later the child began to choke, and it seemed as though tracheotomy would have to be performed, but pressure with the finger on the walls of the abscess from time to time, surrounding the infant with an atmosphere containing steam, and free stimulation, proved eventually to be all that was necessary for its recovery.

The next case was one of an infant thirteen months old, always strong and healthy, who had an attack of acute rhinitis for several days. The rhinitis apparently caused considerable swelling and occlusion of the nares, and the infant after four or five days began to hold its mouth open when breathing and to have difficulty in swallowing. This difficulty in deglutition increased, and it was then noticed that her head was held back. On examining the throat a tense fluctuating swelling was detected in the posterior wall of the pharynx very nearly in the median line. This swelling was incised, a considerable amount of pus was evacuated, and the infant immediately began to breathe more easily and was able to swallow without difficulty. During the next twenty-four hours the abscess filled with pus a number of times, and the pus had to be emptied by pressure with the finger. The infant made a perfect recovery, and had no return of the disease.

In an ordinary case, when the pus has come from the breaking down of glandular material in the posterior wall of the pharynx and already contains pyogenic bacteria, the dangers from reinfection from a wound in the mouth are not serious. But when the pus has come from a tubercular focus in the cervical vertebræ and contains no other organisms than the tubercle bacillus, and is, moreover, in direct communication with an active pathological process in the bone, the risks of a secondary septic infection are considerable. It is, therefore, the practice of many surgeons, nothwithstanding the difficulties of the operation, to attempt to reach the abscess by a careful dissection from the outside of the neck, as in this way t is far easier to keep the wound aseptic. If there is any sign of the abscess ointing externally, the external operation should always be preferred.

In the following case the retropharyngeal abscess was secondary to ervical spondylitis.

FIG. 141.



Retropharyngeal abscess secondary to cervical spondylitis.

This child was being treated for cervical spondylitis, when in addition to the drawing back of its head, as shown in Fig. 141, it began to have increased difficulty in swallowing and to breathe with its mouth open.

On examining the pharynx a bulging, tense, fluctuating abscess of moderate size was detected.

DIVISION X.

DISEASES OF THE LARYNX, TRACHEA, BRONCHI, LUNGS, AND PLEURA.

DISEASES OF THE LARYNX.

THE affections of the larynx which occur most commonly in infants and in young children are neuroses, new growths, lesions produced by foreign bodies, œdema, and laryngitis.

LARYNGOSPASMUS.

Laryngospasmus (*laryngismus stridulus*) is a neurosis which especially affects the larynx in infancy and childhood, and will be described under the name of laryngospasmus when speaking of reflex irritation of the larynx on page 944.

There is a form of laryngospasmus known as *congenital stridor*, characterized by a croaking sound accompanying inspiration, sometimes by a crowing sound. There is no dyspnœa, cyanosis, or diminution in the power of the child to cry or to cough. Expiration is usually noiseless. The stridor is apt to increase in loudness during the first few months of life, but gradually disappears during the second year. Thomson and Turner have concluded from a careful anatomical study that the sound is due to a valvular action of the upper aperture of the larynx by which its lateral walls are drawn inward during inspiration. This is due, not to a congenital malformation, but to an incoördination and to spasmodic action of the muscles of respiration, which they call a choreiform, respiratory spasm, analagous to stammering, and differing from a laryngeal spasm.

NEW GROWTHS.

New growths in the larynx in infants and in children are rare. They may be congenital, but these are very uncommon. They may be malignant, such as epitheliomata and sarcomata, or benign, such as fibromata, myxomata, and papillomata. Those of the former class are so rare that they need here only be referred to. Of the latter class the fibromata and myxomata are too rare to be more than mentioned. The papillomata, on the other hand, although rare, are the most common laryngeal growths in early life. They may produce such serious results that it is important to recognize them at once. They may be congenital. Their cause is not known. Papilloma of the larynx in young children is usually multiple.

The symptoms of this growth appear at about the first, second, or third year. The first symptom that is noticed is hoarseness. This hoarseness, instead of passing off in a few days, as is common when it arises from other affections of the larynx, continues and grows more marked. The next symptom is dyspnœa. This appears at intervals of a few months, or may not arise for some years after the first alteration of the voice. The dyspnœa first appears at night, when the child is asleep. In the daytime, when the child is awake and running about, it may breathe freely. As the papillomata increase in size, the dyspnœa appears in the daytime also, especially when the child makes any exertion. When it is awake and is quiet the breathing may not be noticeably affected, even after the growth has attained a large size. Cough may be present. Usually there is no pain nor difficulty in swallowing. When a child presents these symptoms a careful laryngoscopic examination should be made at once, as in this way only can the diagnosis be verified.

Prognosis.—The prognosis in these cases is bad unless the growths are removed.

Treatment.—The best treatment of multiple papillomata is to remove the growths through the mouth. This is, however, a difficult operation. Without an anæsthetic it is hard to control the child, and many attempts may be required. With an anæsthetic, if the growth is sufficient to cause much obstruction there may be cessation of breathing during the operation, requiring immediate tracheotomy. Many operators prefer to do a previous tracheotomy, especially as it may require more than one operation to clear the larynx. Intubation has been recommended, but it is attended with the danger of breaking off some of the growth which may find its way into a bronchus.

The difficulty of removal is in some cases so great that some of the most competent operators have preferred, if the case is not urgent, to postpone the operation until the child is older. The child during this time must be kept under strict supervision, but local applications are not indicated. These growths, even when completely removed, have a tendency to recur.

FOREIGN BODIES.

Foreign bodies rarely lodge in the larynx, but this accident occurs more commonly in children than in adults, as children are apt to put articles of every description into their mouths.

The symptoms which indicate the presence of a foreign body in the larynx are a sudden attack of suffocation and a change in the sound of the voice in a child who has previously shown no signs of obstruction and no symptoms of laryngeal disease.

The accident is one which is so serious that the child should be placed at once in the hands of a laryngologist. The larynx should be examined with the laryngoscope, and the foreign body removed, if possible, with the forceps. Great care should be taken not to push the foreign body into the trachea, as tracheotomy would then be necessary. For the same reason it is inadvisable to introduce the finger blindly into the larynx, or to do anything which may cause a sudden inspiration.

ŒDEMA.

Œdema of the larynx is not a common condition in early life. It may arise from a number of causes, and is secondary to some disease elsewhere or to some local irritation. It occurs as a rare complication in nephritis and in the acute exanthemata. It may arise from irritation produced by local lesions, such as ulcerations, from foreign bodies, from inhalations of hot vapors, from the swallowing of corrosive liquids, and also as the result of any acute inflammation, such as erysipelas. The diagnosis, as a rule, must be verified by a laryngoscopic examination.

Treatment.—The treatment is that of the disease or local irritation which is causing the œdema. The local application of cold, and, if necessary, scarification of the œdematous tissue, are indicated. If the attack is pronounced and suffocation is imminent, we should be prepared to perform tracheotomy or intubation.

LARYNGITIS.

The most common inflammatory lesions of the larynx which occur in early life are (1) catarrhal laryngitis (false croup, croup) and (2) pseudomembranous laryngitis (membranous croup).

Acute Catarrhal Laryngitis (*false croup*).—The pathological condition which is present in the acute form of laryngitis is a redness or hyperæmia of the laryngeal mucous membrane, accompanied by more or less swelling and serous exudation. The cause of acute catarrhal laryngitis is often a simple extension of a catarrhal condition of the nose and pharynx to the larynx. More rarely a catarrhal condition of the bronchi and trachea may extend upward and involve the larynx. At times the condition appears to be the result of atmospheric changes and undue exposure to dampness and cold. The lumen of the larynx in infancy and in early childhood is so small that even a moderate swelling of the laryngeal mucous membrane may produce sufficient stenosis to give rise to marked obstructive symptoms.

Symptoms.—The symptoms of acute laryngitis are a heightened temperature, 38.3°, 38.8°, 39.4° C. (101°, 102°, 103° F.), and even higher, hoarseness and cough. These symptoms, occurring in connection with a preceding rhinitis or pharyngitis, or arising from a primary inflammation of the larynx, may continue for a number of days without any more serious manifestations, and if the child is kept in an equable temperature the attack may pass off within a week. In some cases, however, another set of symptoms may appear after the primary manifestations have lasted for a variable period. The child may be as well as usual in the morning. Towards the latter part of the day its voice becomes hoarse and there is a slight cough, but without any other symptoms of special importance. After being restless for a time it suddenly awakes, and springs up in bed frightened, often clutching at its throat as if it had a sensation of suffocation. The cough, which during the day was hoarse and somewhat metallic, is now loud and rasping. The child has difficulty in breathing, amounting to orthopnœa, and its face is congested. These symptoms continue for a variable period; usually they last for only one or two hours, but rarely may continue for many hours. In one very uncommon case which was under my care the attack lasted for three or four weeks, during which time it often seemed as though suffocation was imminent. There was in this case no evidence of any lesion beyond a catarrhal laryngitis, and recovery finally took place. These attacks are partly due to obstruction in the larynx from the swollen mucous membrane, the seat of the swelling in these cases being subglottic, but they are largely the result of a neurosis due to a highly sensitive condition of the mucous membrane. On the following day the hoarseness may continue, but the child seems bright and plays about as usual. It is very common for the attack to recur on the second night with greater severity, but in certain cases one attack terminates the disease, and after a variable number of days, the voice becoming clearer each day and the temperature returning to normal, the child recovers. Children who have once had attacks of this kind are liable to have recurrences until they grow older.

Diagnosis.—The diagnosis of acute catarrhal laryngitis is to be made from foreign bodies in the larynx, traumata, and membranous laryngitis. The symptoms in the first two are not preceded by catarrhal symptoms elsewhere, which are almost always met with in catarrhal laryngitis. In a typical case of acute catarrhal laryngitis with suffocative symptoms the diagnosis is not difficult. The acute, sudden onset of the attack in the night, the loud, metallic cough, and the heightened temperature, are distinct from the moderate temperature and the slow, progressive stenosis caused by the formation of a membrane in the larynx.

Prognosis.—Acute laryngitis is a self-limited disease, and one in which the prognosis is almost invariably good. In children who are very weak and debilitated the interference with their respiration may prove to be serious, but these cases are rare and should be treated with stimulants until the disease has run its course. The symptoms of acute catarrhal laryngitis are so terrifying to the parents that the physician is often led to look upon the disease more seriously than is necessary.

Treatment.—The treatment of acute catarrhal laryngitis is to keep the child in a room of an equable temperature of about 20° to 21° C. (68° to 70° F.) until its temperature has become normal and the hoarseness has disappeared. I have also found that a few drops of wine of ipecac, given in the latter part of the afternoon and just as the child is going to sleep, are of benefit in preventing the spasmodic, obstructive symptoms which have just been described as occurring in the night. When the attack occurs in the night the symptoms of suffocation can be best relieved by a dose of from five to ten minims of wine of ipecac, or an amount sufficient to nauseate slightly. An emetic will sometimes cut short an attack of this nature, but in many cases is not necessary. An amount of ipecac sufficient to nauseate slightly, but not to cause the child to vomit, will often so relax the spasm of the larynx that the attack will soon be relieved. In many cases, however, even if vomiting has been produced, the attack continues, and other measures for relief are required. In addition to the ipecac, moderate doses of tinctura opii camphorata, 0.3 to 0.6 c.c. (5 to 10 minims), may be given. An atmosphere of steam usually gives great relief to the spasm. Many accidents have occurred from the improper management of the steam and from giving such emetics as turpeth mineral. The necessity for operative measures rarely arises.

Chronic Laryngitis.—A chronic form of laryngitis occurs in both infants and children. Syphilitic infants are at times affected by chronic laryngitis. It may also occur in tubercular disease, but is not common. When an acute laryngitis has occurred a number of times, or when an attack has been much prolonged by improper treatment, chronic laryngitis may result. In many of these cases the voice, on the slightest exposure to dampness, becomes hoarse, and this hoarseness, after a time, may be continuous.

TREATMENT.—The treatment is to apply astringents to the pharynx, which is almost universally involved, and to regulate the climatic surroundings of the child. Local applications to the larynx in these cases are seldom necessary.

Pseudo-Membranous Laryngitis.—A pseudo-membrane in the larynx may be caused by the inhalation of irritating vapors, or by the inspiration of corrosive liquids. These accidents are so readily recognized that there is no difficulty in determining the cause of the pseudo-membrane in these cases. Treatment for the relief of the stenosis should be instituted at once. This consists in the application of cold and such soothing inhalations as 3.75 c.c. (1 drachm) of compound tincture of benzoin in a quart of boiling water. The complicating œdema which is often present in these cases may require operative interference.

The most common cause of pseudo-membranous laryngitis, and the one which probably in nearly all cases produces it, is some form of micro-organism. These micro-organisms may be of several varieties. Until it is proved not to be so, however, pseudo-membranous laryngitis must be looked upon clinically as infectious and due most commonly to the Klebs-Loeffler bacillus. There is no doubt but that a simple catarrhal inflammation localized in the larynx may be produced by the Klebs-Loeffler bacillus. The symptoms, diagnosis, and treatment of laryngeal diphtheria have been described on page 465.

Some aid in the differential diagnosis of pseudo-membranous from acute catarrhal laryngitis can be obtained from the temperature, which in the latter is considerably raised, while in the former it is moderate and sometimes normal or subnormal. The slow course of a constitutional disease gradually causing obstruction is significant of this infectious form of laryngitis.

DISEASES OF THE TRACHEA.

Pathological conditions of the trachea not connected with those of the air-passage above or below it are uncommon. The lesions of the trachea may be primary or secondary. In the latter they are merely an extension of the disease from the larynx or the bronchi, and do not play an especially significant part in the attack. The only primary disease of the trachea which is common in infancy and childhood is an acute inflammation occurring in its mucous lining. When this inflammatory condition is present, it produces an irritating cough which can usually be excited by gentle pressure over the trachea,—about the only method by which we can locate the disturbance.

The treatment is to protect the child from an atmosphere which is either too hot or too cold, from high winds, and from dust. Douching the front of the neck with cold water several times during the day is also desirable. Direct applications to the trachea are almost impossible in young children.

DISEASES OF THE BRONCHI AND LUNGS.

The diseases which affect the lungs in infancy and childhood differ somewhat from the same diseases occurring in later life, on account of the differences which exist in the anatomical conditions at birth and during the early years of life, especially the first five. The principle differences are that the bronchi occupy a relatively larger portion of the lung in the child than in the adult, that in the former the interstitial tissue is present in a larger amount, that the cavities of the air-vesicles are smaller, and that their walls are relatively thicker; also that the epithelial cells lining the air-vesicles are very numerous. These cells in inflammation tend to rapid cell-division, which is one of the characteristics that mark the pneumonia of childhood. These anatomical differences are of great significance when any part of the lung is diseased, and tend to make a congested lung of much more serious import in the young child than in the adult. Post-mortem examinations often show various lesions which during life were not represented by any definite symptoms, so that we cannot expect the clinical diagnosis to include entirely the pathological lesions.

bacillus. The symptoms, diagnosis, and treatment of laryngeal diphtheria have been described on page 465.

Some aid in the differential diagnosis of pseudo-membranous from acute catarrhal laryngitis can be obtained from the temperature, which in the latter is considerably raised, while in the former it is moderate and sometimes normal or subnormal. The slow course of a constitutional disease gradually causing obstruction is significant of this infectious form of laryngitis.

DISEASES OF THE TRACHEA.

Pathological conditions of the trachea not connected with those of the air-passage above or below it are uncommon. The lesions of the trachea may be primary or secondary. In the latter they are merely an extension of the disease from the larynx or the bronchi, and do not play an especially significant part in the attack. The only primary disease of the trachea which is common in infancy and childhood is an acute inflammation occurring in its mucous lining. When this inflammatory condition is present, it produces an irritating cough which can usually be excited by gentle pressure over the trachea,—about the only method by which we can locate the disturbance.

The treatment is to protect the child from an atmosphere which is either too hot or too cold, from high winds, and from dust. Douching the front of the neck with cold water several times during the day is also desirable. Direct applications to the trachea are almost impossible in young children.

DISEASES OF THE BRONCHI AND LUNGS.

The diseases which affect the lungs in infancy and childhood differ somewhat from the same diseases occurring in later life, on account of the differences which exist in the anatomical conditions at birth and during the early years of life, especially the first five. The principle differences are that the bronchi occupy a relatively larger portion of the lung in the child than in the adult, that in the former the interstitial tissue is present in a larger amount, that the cavities of the air-vesicles are smaller, and that their walls are relatively thicker; also that the epithelial cells lining the air-vesicles are very numerous. These cells in inflammation tend to rapid cell-division, which is one of the characteristics that mark the pneumonia of childhood. These anatomical differences are of great significance when any part of the lung is diseased, and tend to make a congested lung of much more serious import in the young child than in the adult. Post-mortem examinations often show various lesions which during life were not represented by any definite symptoms, so that we cannot expect the clinical diagnosis to include entirely the pathological lesions.

for, especially if after from twenty-four to forty-eight hours the temperature does not fall to 37.7° or 38.3° C. (100° or 101° F.)

After a few days the severity of the symptoms lessens, the cough becomes looser, the râles gradually disappear, and under favorable conditions the symptoms subside entirely in a week or ten days. There is seldom any expectoration in children under six or seven years. In the more severe cases the râles are more numerous than in the mild form of the disease, but are of the same character. In the course of some cases of bronchitis a temporary localized diminution or even absence of the respiratory sound may result from the occlusion of a bronchus. This is especially common in infants, and ordinarily is not accompanied by a change in the percussion-sound. This form of bronchitis is the one which affects the larger and the medium-sized bronchi.

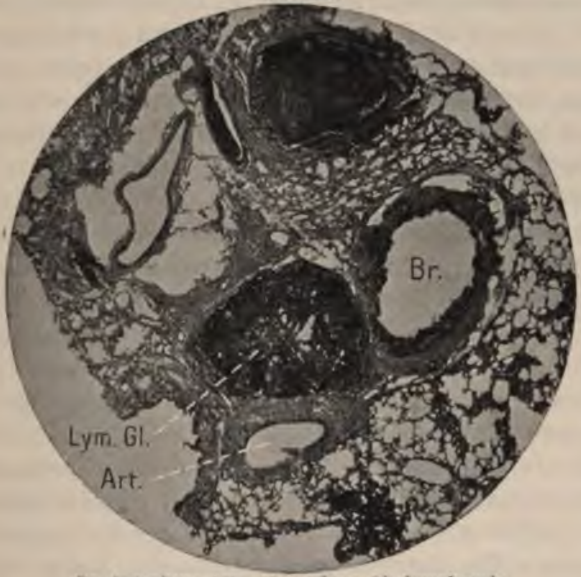
There is no characteristic temperature in bronchitis. As a rule, it is moderate, 37.2° to 38.3° C. (99° to 101° F.) but it varies greatly according to the individual and to the degree of nervous excitement.

The blood shows nothing of diagnostic value (Cabot). Leucocytosis is not present in the usual forms of acute bronchitis, but only in the so-called capillary bronchitis (broncho-pneumonia), in which a leucocytosis is usually present.

Diagnosis.—The diagnosis of the ordinary cases of acute bronchitis, in which only the large and medium-sized bronchi are affected, is not difficult, the only disease for which it is likely to be mistaken being broncho-pneumonia. In this latter disease the greater severity of the symptoms and the higher temperature will usually show its presence, even though the physical signs may be only those described as occurring in bronchitis. In the more severe forms of bronchitis it is sometimes exceedingly difficult to make the differential diagnosis from broncho-pneumonia. If, however, the temperature, after three or four days, remains high, and rises to 39.1° or 39.4° C. (102.5° or 103° F.), with marked remissions and exacerbations, the diagnosis becomes doubtful, and in these cases we should strongly suspect that a broncho-pneumonia has arisen as a complication. We must, however, remember that in certain cases of broncho-pneumonia the temperature may be as moderate as in acute bronchitis, and we must therefore rely on a combination of symptoms rather than on any one symptom or sign. An important point in the differential diagnosis between bronchitis and broncho-pneumonia is that the physical signs in the former are much more frequently found in all parts of the thorax, while in the latter circumscribed groups of râles are often detected in different parts of the lungs. The râles in themselves, however, are not distinctive, as the râles in broncho-pneumonia are mostly those of the accompanying bronchitis. Although the physical signs of dulness and bronchial respiration are conclusive evidences that the case is not one of bronchitis alone, yet an absence of these signs does not justify us in excluding broncho-pneumonia. When the dyspnœa, general prostration, bronchitis. The specimen shows hyperplasia of the lymph-nodes due to bronchitis. This condition is very commonly found in bronchitis, especially when it occurs in debilitated children. There is desquamation of the epithelium lining the bronchi, as well as a slight thickening of their walls.

SYMPTOMS.—The onset of acute bronchitis is usually mild, but I have seen in a debilitated infant a simple, uncomplicated bronchitis begin with a convulsion. The symptoms are very variable in their intensity, and are usually more acute and definite in a previously healthy child than in debilitated children, in whom they are often subacute and of an insidious nature. In infants and young children the bronchitis is almost always preceded by a catarrhal condition of the upper air-passages. In the mild cases there is an elevation of the temperature, 37.7° to 38.3° C. (100° to 101° F.), cough of greater or less severity, and a slight lessening of the appetite. On physical examination the pulmonary resonance is found to be normal. A few sibilant and sonorous râles are heard with especial frequency in the area between the scapula and the vertebral column. Moist râles may also be heard. In severe cases the children suffer from more or less discomfort, produced probably by the thoracic pain, although in young children the locality of the pain cannot, as a rule, be determined. The cough is hard and dry, the respirations may be slighly raised, and the pulse accelerated. The children may appear quite ill for two or three days, and the temperature may rise as high as 38.8° or 39.1° C. (102° or 102.5° F.); but when this latter point is reached the onset of a broncho-pneumonia should be carefully watched

FIG. 142.



Br., bronchus; Art., artery; Lym. Gl., lymph-node.

for, especially if after from twenty-four to forty-eight hours the temperature does not fall to 37.7° or 38.3° C. (100° or 101° F.)

After a few days the severity of the symptoms lessens, the cough becomes looser, the râles gradually disappear, and under favorable conditions the symptoms subside entirely in a week or ten days. There is seldom any expectoration in children under six or seven years. In the more severe cases the râles are more numerous than in the mild form of the disease, but are of the same character. In the course of some cases of bronchitis a temporary localized diminution or even absence of the respiratory sound may result from the occlusion of a bronchus. This is especially common in infants, and ordinarily is not accompanied by a change in the percussion-sound. This form of bronchitis is the one which affects the larger and the medium-sized bronchi.

There is no characteristic temperature in bronchitis. As a rule, it is moderate, 37.2° to 38.3° C. (99° to 101° F.) but it varies greatly according to the individual and to the degree of nervous excitement.

The blood shows nothing of diagnostic value (Cabot). Leucocytosis is not present in the usual forms of acute bronchitis, but only in the so-called capillary bronchitis (broncho-pneumonia), in which a leucocytosis is usually present.

Diagnosis.—The diagnosis of the ordinary cases of acute bronchitis, in which only the large and medium-sized bronchi are affected, is not difficult, the only disease for which it is likely to be mistaken being broncho-pneumonia. In this latter disease the greater severity of the symptoms and the higher temperature will usually show its presence, even though the physical signs may be only those described as occurring in bronchitis. In the more severe forms of bronchitis it is sometimes exceedingly difficult to make the differential diagnosis from broncho-pneumonia. If, however, the temperature, after three or four days, remains high, and rises to 39.1° or 39.4° C. (102.5° or 103° F.), with marked remissions and exacerbations, the diagnosis becomes doubtful, and in these cases we should strongly suspect that a broncho-pneumonia has arisen as a complication. We must, however, remember that in certain cases of broncho-pneumonia the temperature may be as moderate as in acute bronchitis, and we must therefore rely on a combination of symptoms rather than on any one symptom or sign. An important point in the differential diagnosis between bronchitis and broncho-pneumonia is that the physical signs in the former are much more frequently found in all parts of the thorax, while in the latter circumscribed groups of râles are often detected in different parts of the lungs. The râles in themselves, however, are not distinctive, as the râles in broncho-pneumonia are mostly those of the accompanying bronchitis. Although the physical signs of dulness and bronchial respiration are conclusive evidences that the case is not one of bronchitis alone, yet an absence of these signs does not justify us in excluding broncho-pneumonia. When the dyspnœa, general prostration, and restlessness are slight and the temperature moderate, the case is likely to be one of bronchitis, while if these symptoms are marked, and are combined with cyanosis, dilatation of the alæ nasi, and a higher temperature, at least a provisional diagnosis of broncho-pneumonia should be made. In some cases the differential diagnosis will also have to be made from the onset of a pleuritis or of a lobar pneumonia, but the moderate temperature and respirations, the normal percussion-sounds, and the diffuse bilateral râles in bronchitis usually make the diagnosis from these diseases quite evident. If the blood-count shows an absence of leucocytosis it favors the diagnosis of bronchitis.

PROGNOSIS.—The prognosis, when no complication arises and the child is previously healthy, is good. In debilitated children, and especially when rhachitis is present, even a mild form of bronchitis may prove to be serious, on account of the danger of a complicating broncho-pneumonia, and in these cases the prognosis is much more unfavorable.

TREATMENT.—The treatment of acute bronchitis is essentially hygienic. The child should be confined to a warm, well-ventilated room which has a sunny exposure, and which is heated by an open fire to a temperature of about 20° to 21.1° C. (68° to 70° F.). A few minims of wine of ipecac should be given if the cough is unusually dry, and to this a few minims of tinctura opii camphorata may be added if the patient is very nervous. These remedies are all that will usually be needed in an attack of acute bronchitis. When a rhachitic child or one who is much debilitated is attacked by the disease, especial care must be taken to support its strength by stimulants and food.

Unusual Form of Acute Bronchitis.—Besides the acute bronchitis just described, I have met with a class of cases which are extremely rare, but which, apparently, are instances of an exacerbation of an ordinary bronchitis through the involvement of the smaller bronchi, *not the terminal ones.* I have seen only six or eight of these cases. These, from their clinical history, seem to have been cases of bronchitis rather than of broncho-pneumonia. I speak of them separately, as the symptoms differ somewhat from those of an ordinary bronchitis. This form of bronchitis has no connection with that which was formerly erroneously called capillary bronchitis, but which is well known to be only an early stage of broncho-pneumonia. This form of bronchitis in my cases has commonly occurred in infants in the first two years of life, though I have met with it as late as the third year. The cause, so far as could be ascertained, was the same as in an ordinary bronchitis, a catarrhal condition of the upper air-passages usually preceding the attack. The onset of the disease was rapid, and the symptoms soon became very severe. The temperature was, as a rule, moderately raised, 37.7° to 38.3° C. (100° to 101° F.). The cough was continuous, and dyspnœa, with more or less cyanosis, rapidly developed. An examination showed normal resonance throughout the whole thorax, and fine moist râles. The respirations were ever it is exposed to a damp atmosphere, a loud wheezing will be heard in the chest. Auscultation will reveal the presence of sonorous râles everywhere, and in this variety, as well as in other forms of chronic bronchitis, a roughened sensation may sometimes be felt on palpation during respiration.

DIAGNOSIS.—The differential diagnosis is to be made from chronic affections of the lungs in which the thickening of the interstitial tissue has taken place with a resulting diminution of resonance, and from the condition in which the bronchi are dilated. In the latter case there are accompanying symptoms of a profuse exudation of purulent matter.

Chronic bronchitis is also to be distinguished from reflex coughs of a nervous character, unaccompanied by any disease of the respiratory tract. Causes for such reflex coughs may be found in irritation of the pharynx and ear, from chronic gastric indigestion, from pulmonary congestion, from the disturbances in circulation due to cardiac disease, and, finally, from pressure upon the bronchi of enlarged mediastinal glands.

PROGNOSIS.—The prognosis of chronic bronchitis varies according to the cause. When it is secondary to disease of some other organ, it depends entirely upon the prognosis of that disease. In rhachitic children the prognosis is unfavorable, and in them a broncho-pneumonia is especially liable to develop, with a fatal issue. Cases of chronic bronchitis are also liable to be invaded by the tubercle bacillus. In cases which are the result of acute bronchitis in individuals otherwise healthy, the prognosis is favorable, provided the proper treatment can be carried out. As emphysema in chronic bronchitis is rare in children in comparison with adults, the chances for recovery in the former are correspondingly good.

TREATMENT.—The treatment of chronic bronchitis is essentially climatic. The children should be kept in a warm dry climate for some months after the bronchitis has entirely disappeared. Especial care should be taken that the child is suitably protected by flannel undergarments. When other treatment is required, as a rule, tonics will prove of more benefit than the drugs which are usually administered for their direct effect upon the bronchial mucous membrane.

FIBRINOUS BRONCHITIS.

During the course of what may appear to be an ordinary bronchitis, in rare instances a fibrinous form of bronchitis has been met with. In this variety masses of fibrin in the bronchi form casts of various extent according to the number of the bronchi which are affected. Similar fibrinous casts of the bronchi may be found in cases of diphtheria of the larynx by direct extension to the bronchi, but do not represent fibrinous bronchitis.

The disease may run a short course of days or weeks, but is usually chronic and may last for years. The paroxysms may also be periodic.

The symptoms and course of all these cases are very similar, so that I shall speak only of one other child, whom I saw in consultation.

A male, seven months old, and previously healthy, for two days had a slight cough, with a few sonorous râles in the chest and a temperature varying from 36.6° to 37.2° C. (98° to 99° F.). On the third day of the attack he was suddenly seized with increased cough, dyspnœa, cyanosis, respirations of 70, a pulse of 160, and a temperature of 38.3° C. (101° F.). An examination of the chest showed normal resonance and fine moist râles throughout both lungs. The infant was treated with aromatic spirit of ammonia and brandy in alternate doses. On the following day the temperature fell to 37° C. (98.6° F.), and the fine râles were replaced by coarse and sonorous râles. The bronchitis lasted for a few days, and the infant then recovered entirely.

CHRONIC BRONCHITIS.

Etiology.—Chronic bronchitis may result from a series of attacks of acute bronchitis, or from a number of other causes. Among these may be mentioned various affections of the lungs, such diseases connected with malnutrition as rhachitis, and prolonged attacks of pertussis.

Pathology.—The pathological conditions occurring in chronic bronchitis vary greatly in degree, and the lesions found at the post-mortem examination are often slight in comparison with the severity of the symptoms during life. In most cases there is a considerable production of mucus, pus, and serum. In cases which have lasted for a long time, in addition to the inflammatory products affecting the walls of the bronchi there may be dilatation of one or more bronchi, and the muscular coat may be thickened or thinned. Emphysema may also result.

Symptoms.—The symptoms of chronic bronchitis are very much the same as those of acute bronchitis, except that the temperature is not so apt to be heightened, while the general symptoms of malaise, anorexia, and loss of weight are more prominent. In severe and prolonged cases in which emphysema is present, the thorax may assume the position of full inspiration, the ribs being permanently raised and the antero-posterior diameter of the chest increased. The physical signs are the same as in acute bronchitis, so far as the râles are concerned. The resonance is usually normal except when the chronic process has produced emphysema, in which case there are areas of hyper-resonance often associated with a tympanitic tone. Occasionally atelectasis of considerable areas of the lungs may take place, with a resulting diminution of the respiratory sound. This occurrence may in some cases prove to be serious, but in others the accompanying symptoms are mild, and the alveoli may again return to their normal degree of inflation.

There is one form of bronchitis which from its duration may be called chronic, and yet which from the very slight degree of constitutional symptoms that accompany it corresponds rather to a subacute affection. In these cases, which usually occur in infancy and in early childhood, the child often appears quite well, but for long periods of weeks, or when-especially of the bronchi, commonly precedes the attack for some days. The onset is usually sudden, and generally occurs at night. The child is seized with distressing dyspnœa, mainly expiratory, the respiration being accompanied by a wheezing sound. The face is anxious, and if the attack continues for some time it becomes slightly cyanotic. The respirations are not especially increased in frequency. The pulse is rapid, and when the dyspnœa is very intense becomes weak. The temperature is not raised, and when the paroxysm is prolonged it may become subnormal. The physical signs are mostly diffuse, sibilant, and sonorous râles, rhonchi, and muscular rumbles, which obscure or render inaudible the respiratory murmur. The attack may last for a number of hours, or even for days. The paroxysms vary in their severity, and, as a rule, are followed by considerable exhaustion. The frequency of the attacks varies; they may occur often or only at intervals of months.

Diagnosis.—Attacks of asthma are generally easily diagnosticated by the spasm, the history of previous attacks, and the characteristic sputum and blood. Cardiac, renal, and thyroid dyspnœa—that is, conditions known as "false asthma"—can be excluded by the examination of the blood and sputum and by the recognition of the respective diseases.

Mechanical irritation of the bronchi, as by pressure of an aneurism or of enlarged glands, may bring on a spasm of the bronchioles resembling true asthma, and here again the examination of the blood and sputum are of great service.

Spasm of the glottis produces a noisy dyspnœa, but the difficulty is with inspiration instead of expiration. The sound produced is more of a crow than a wheeze, and no râles can be heard in the lungs.

Prognosis.—The prognosis of asthma with regard to the especial attack is good. When the disease is not hereditary the children very commonly recover from it as they approach the age of puberty. In many cases the attacks seem to depend upon some local affection of the air-passages, and the cure of these local lesions will often be followed by recovery from the attacks of asthma.

Treatment.—In the treatment of asthma, the nose and throat should be carefully examined for local diseases, as the attacks may be caused by the different forms of rhinitis, adenoid growths, or enlarged tonsils. The children should be protected from unfavorable atmospheric influences, a high, dry, inland air usually being better suited to them than sea-air. In some cases, especially of a mild form, the fumes of nitre paper will give considerable relief. In very severe attacks hydrate of chloral may be given, either by the mouth or by enemata. Antispasmodics, such as belladonna and lobelia, can also be used. There is no one drug which will relieve the paroxysms of asthma except morphine, which should be used with great caution. Iodide of potassium in gradually increasing doses is in some cases beneficial. Especial attention should be paid to the general hygiene and to the diet of the child.

PNEUMONIA.

Pneumonia is an acute or subacute affection of the lungs, due to the action of infectious micro-organisms, the pathology, symptoms, and prognosis of which vary according to the particular bacterium which is the exciting cause of the inflammation.

Two general types of the disease are usually described, according to the mode of invasion and to the distribution of the pathological lesions; (1) *lobar pneumonia*, in which the whole or greater part of a lobe of the lung is involved, and (2) *broncho-pneumonia*, in which individual lobules of the lung are affected, forming small areas of consolidation irregularly distributed.

One particular organism, the *pneumococcus of Fraenkel*, shows a special tendency to cause an inflammation of a lobar type, and such a disease, accurately speaking, should be termed "*pneumococcus lobar pneumonia*." It is this condition which is generally referred to when the terms "*lobar pneumonia*," "*acute croupous pneumonia*," or "*acute fibrinous pneumonia*" are used.

Broncho-pneumonia, or *lobular pneumonia*, is, on the other hand, due to a number of different organisms, which will be more fully described later. The especial point to be emphasized in connection with the subject of classification is the characteristic tendency of these organisms to produce an interstitial inflammation of the peribronchial tissues of the terminal bronchi, with exudation into the neighboring alveoli and consequent consolidation of the lobules of the lungs. Such areas of consolidation may occasionally, by confluence, merge into one large mass, and end by producing a consolidation of the whole of a lobe, giving rise to physical signs and sometimes to macroscopic appearances which are essentially the same as a lobar pneumonia, but all cases of this variety will, on microscopic examination, show the appearances of a lobular infection differing in many respects from the microscopic pathological appearances of a pneumococcus lobar pneumonia. Tuberculosis of the lung is essentially a broncho-pneumonia, but with pathological and clinical features which are so distinctive that it is more convenient to describe it in connection with the general subject of tuberculosis (page 393).

Both the lobar and the lobular or broncho-pneumonias are diseases due to infectious micro-organisms. In view, however, of the variety of etiological factors in a broncho-pneumonia, and of the occasional cases which bear so close a resemblance to a lobar pneumonia, and inasmuch as the specific organism of pneumococcus pneumonia is also, in a large proportion of cases, the direct cause of a broncho-pneumonia, it is much more convenient for the present to describe these affections together and in connection with the general subject of diseases of the lung. For the same reasons we shall not attempt to isolate the pneumococcus lobar pneumonia and to classify it among the specific infectious diseases.

PNEUMOCOCCUS LOBAR PNEUMONIA.

Pneumococcus lobar pneumonia is an acute infectious, self-limited disease, characterized in typical cases by sudden onset, high fever, consolidation of one or more lobes by a fibrinous exudation into the alveoli, marked leucocytosis, running a course in children of from five to eight days and ending abruptly by crisis.

Etiology.—Acute infections of the lungs, which are associated with a consolidation of the whole or greater part of a lobe of a lung, are due in a great majority of cases, probably in as many as 95 per cent., to the action of the *pneumococcus of Fraenkel*, which has also been called the *micrococcus lanceolatus*. This organism is, at times, also a direct cause of pleurisy, pericarditis, endocarditis, peritonitis, and meningitis.

The disease is almost invariably primary. It may appear in an epidemic form and run a more fatal course than in sporadic cases. It may occur at any age, but in the first two years of life it is rare in comparison with broncho-pneumonia, which is the usual form of pneumonia in infancy. In the third year it becomes more common, and, according to Osler, increases in frequency up to the sixth year, when it diminishes up to the fifteenth year. The proportion of cases then again begins to rise. Recurrent attacks of pneumococcus lobar pneumonia are not so frequent in young children as in adults.

Exposure to cold and especially to sudden atmospheric changes seems to render the individual more susceptible to the invasion of the micro-organism. When this form of pneumonia occurs, as it sometimes does, as a secondary infection in such diseases as pertussis, measles, and scarlet fever, it is usually in children over three years of age (Holt). These secondary pneumonias are, however, much more likely to be in the nature of broncho-pneumonia.

Pathology.—The pathological condition which occurs in lobar pneumonia is an acute exudative inflammation which involves progressively the whole of one lobe, or the larger part of one lung, or portions of both lungs. There is no especial distinction between the lesions of lobar pneumonia as they occur in children and those which are met with in adults, except so far as the anatomical conditions differ according to the age of the individual. It is much more common, however, to have the pneumonic process begin in the apices of the lungs in children than in adults. Holt's statistics show that both lungs are affected with about the same frequency, and that the order of frequency is left base, right apex, right base, left apex.

The stages of *congestion*, *red hepatization*, *gray hepatization*, and *resolution* take place in succession in the pneumonia of the child as in that of the adult. In the stage of congestion the lung is hyperæmic and œdematous and the air-vesicles contain fibrin, pus, granular matter, red blood-cells and epithelial cells. The epithelium of the air-vesicles is swollen, and there are large numbers of white blood-cells in the capillaries. The large bronchi are congested. The small bronchi contain the same inflammatory products as do the air-vesicles. This stage lasts only a few hours, as a rule, but may be protracted for several days. When the exudation of the inflammatory products has reached its full development the presence of these products within the air-vesicles and bronchi causes the lung to be slightly enlarged, and at this time it is said to be in the condition of red hepatization. After the air-vesicles have become completely filled with exudation there follows a period during which the exudation first becomes decolorized and then degenerated. This is the period of gray hepatization. This happens at a variable time, which is usually shorter in children than in adults. The color finally becomes gray. The exudate then undergoes still further degeneration and softening, and is removed by the lymphatics. This is the stage of resolution. Resolution should begin immediately after defervescence and be completed within a few days, but it may not begin until a number of days after defervescence, and may be unusually protracted.

The bronchi are almost always affected in lobar pneumonia. The pneumonic process may occur in small patches, but usually involves an entire lobe. The lower lobes are the ones which are most frequently affected in early life, but the locality of the pneumonia is of pathological rather than of clinical importance, as the disease may attack any part of the lungs. It is generally a unilateral disease, but in some cases it may be bilateral.

SYMPTOMS.—The onset of a pneumococcus lobar pneumonia is, as a rule, very acute, and in the infant or young child is frequently ushered in by vomiting and sometimes by convulsions. The temperature rises suddenly and remains high, as a rule, until the crisis. The pulse-rate is increased. The child appears profoundly infected. The face is flushed, the skin hot and dry. There is a short, dry, painful cough. The respirations are rapid and shallow. The alæ nasi expand with each inspiration, and the dyspnœa is marked. The mind at first is clear, but as the process continues delirium and stupor assert themselves as prominent symptoms. In from five to seven days the disease in typical cases runs its course. The temperature falls by crisis. Sometimes within a few hours the picture changes suddenly. The breathing becomes easy, the skin moist and then bathed in perspiration, the pulse slow and full, the appetite returns, and convalescence, barring complications, is rapid.

The individual symptoms vary so greatly in different cases that they can be understood best when considered separately.

Onset.—The onset of the disease is preceded by a prodromal stage of a few hours only, sometimes with mild catarrhal symptoms, sometimes with only chilly sensations, headache, and malaise. A definite rigor is very uncommon under five years; convulsions are more frequent in infants and are uncommon at a later period. Vomiting or diarrhœa, *Cough.*—Cough is a common symptom, not only in the beginning of the disease, but also during its whole course, and often seems to be painful. It may, however, be absent for several days in the beginning of the attack. There is rarely any expectoration before the seventh or eighth year, and hence in infants and young children we rarely see the "rusty" sputum so characteristic of pneumococcus pneumonia. When it is obtained it is generally rusty and tenacious, and, on staining, the pneumococci are found within the leucocytes in large numbers.

Blood.—The blood shows a marked increase in the leucocytes, which begins at the time of the chill. In cases in which a pseudocrisis occurs the leucocytosis persists, while, in certain cases, at the time of or a few hours before the true crisis the white blood count begins to diminish, and reaches its normal one or two days after the temperature. If resolution is by lysis, the reduction in the white cells is gradual. In delayed resolution, the leucocytosis persists, sometimes for weeks, especially if abscess, empyema, or gangrene develops as a complication. Cases of pneumonia which show an absence of leucocytosis, except in the very mildest forms, are generally considered of very unfavorable prognosis. A high and increasing white blood count is of value only as an aid to diagnosis, and is no guarantee whatever of a favorable issue.

Urine.—The urine shows the characteristics of a febrile urine,—high color, high specific gravity, and increased acidity, with a slight trace of albumin and pure hyaline and finely granular casts. The chlorides are absent or greatly reduced at the height of the fever owing to the large amount which is deposited in the consolidated lung. At the time of the crisis there is often a marked increase in the amount of the urine, and the chlorides reappear.

Skin.—Herpes of the nose, lips, genitals, or anus occurs not uncommonly in association with pneumococcus lobar pneumonia. Redness of one cheek is an occasional phenomenon, but is not necessarily on the same side as the consolidation. Cyanosis is seen in connection with failing circulation.

Digestive Organs.—The tongue is white, furred, and dry. The appetite fails, diarrhœa or constipation are apt to appear, and meteorism with considerable distension of the abdomen is sometimes a distressing and grave symptom. The spleen is usually slightly enlarged, and its edge is occasionally palpable.

Crisis.—The time when the temperature falls and the crisis takes place varies. It may occur as early as the third or fourth day, but is usually between the fifth and the eighth day. It may, however, be delayed until the ninth or tenth day, and in rare cases still longer. This rapid fall in the temperature, associated with the initial sudden rise, are two of the most typical and valuable symptoms in the diagnosis of a pneumococcus lobar pneumonia. When the temperature falls at the crisis of the disease it is very apt to become subnormal, and to remain so for a number of days. Sometimes after the temperature has fallen to the normal it may rise again, but, as a rule, another rise of temperature points towards the involvement of some fresh area of the lung or to some complication, such as pleurisy. The fall of temperature at the time of the crisis is often accompanied by symptoms of great prostration and even collapse, and it is therefore important to watch carefully for the crisis and to be prepared to combat these symptoms, as death has occurred at this time. The normal height of the temperature in acute lobar pneumonia, according to the extensive observations of Hoit, is from 40° to 40.5° C. (104° to 105° F.). In children over three years of age the temperature curve resembles the adult type in being regular and falling by crisis, while under three years of age the proportion of typical cases is much less, and there is more irregularity in the course of the temperature, which may fall by lysis. The younger the individual the more likelihood there is to be a wide fluctuation in the range of the temperature, which has a tendency to be of the remittent type even in uncomplicated cases. Morrill and others have shown that if the fall in the temperature is prolonged beyond the twelfth day it is apt to subside by lysis.

PHYSICAL SIGNS.—*Inspection.*—The principal features observed on inspection are the flushed cheeks, herpetic vesicles about the mouth or nose, short, rapid, and superficial respiration, dilatation of the alæ nasi, and sometimes deficient expansion on the affected side.

Palpation.—In typical cases the tactile fremitus is much increased. If the air is excluded from the bronchi by secretions or fibrinous plugs, tactile fremitus is not obtained. The obstruction may sometimes be removed by a few hard coughs, and the fremitus then returns.

Percussion.—The percussion-note over an area of consolidation varies from dulness to flatness: it may be tympanitic in quality in the early and late stages of the disease. In the pneumonias of infants and children dulness is especially likely to be absent, and if the base of the lung is consolidated, a tympanitic quality may be obtained by transmission from a distended stomach or colon. Wintrich states that the minimum amount of consolidation, when superficial, which will produce dulness is an area five centimetres in diameter and two centimetres deep.

Auscultation.—The respiration in infants and young children is normally somewhat harsh or broncho-vesicular in character, and should not be mistaken for the tubular or bronchial breathing which is characteristic of a consolidation. If the tubes are filled with mucus and fibrin, the respiratory sounds may be much diminished and even absent, closely simulating a pleural exudation. In the very early stages the breathing may be feeble or suppressed over the affected area, and very fine crackling or crepitant râles may be heard at the end of each inspiration, which disappear during consolidation and reappear at the beginning of resolution. Medium and coarse moist râles are much more common in children than the crepitant râles. The voice-sounds are intensified, seem near to the ear (bronchophony), and sometimes have a nasal quality (egophony).

VARIETIES.—Certain variations in the type of pneumococcus lobar pneumonia occur, and have been designated by especial names. In *apex pneumonia* the inflammation is limited to the apex of the lung. In *massive pneumonia* the exudation fills even the bronchi, completely consolidating the lung, and giving rise to physical signs which can be differentiated from pleural exudation only by exploratory aspiration. In *migratory* or *creeping pneumonia* one lobe after another is involved. *Cerebral pneumonias* are simply those forms which are associated with severe cerebral symptoms. Some writers describe an *abortive type* of pneumococcus pneumonia in which the signs are those of the stage of congestion, and the disease runs a course of from twenty-four to forty-eight hours. I, myself, have not, however, seen in children any cases in which the evidence was sufficient to justify the diagnosis.

Central pneumonia is a type of the disease especially common in children. The clinical symptoms are present, but the physical signs are absent, as the consolidation is deep-seated. The signs may not come to the surface for many days, or resolution may take place before they appear.

Delayed Resolution.—The crisis in lobar pneumonia is not always accompanied by a resolution of the consolidation; the local signs may persist, and disappear only in the course of several weeks. In these cases of delayed resolution the temperature frequently falls by lysis.

The following case illustrates a lobar pneumonia in which resolution was delayed for thirty days.

A girl, four years old, and perfectly well and strong, was suddenly attacked with vomiting, pain in the right side, and cough accompanied, according to her mother, by a reddish-brown sputum. Physical examination on the following day revealed nothing abnormal except a few fine moist râles at the base of the right lung behind. The pulse was 170, the respirations 60, and the temperature 39.4° C. (103° F.). On the following day the temperature still remained raised, and there was dulness on percussion over the lower right lobe behind, with bronchial respiration. On the following day the dulness had extended over the whole of the right lung in front and behind. The temperature varied from 38.8° to 39.4° C. (102° to 103° F.), the pulse from 150 to 160, and the respirations from 50 to 60. These symptoms continued until the eighth day from the onset of the attack, when the temperature was found to be 38.4° C. (101.2° F.), the respirations 48, and the pulse 160. During the next nine days the temperature, pulse, and respirations remained the same, and there was no change in the physical signs of the lung, except that in addition to the dulness and bronchial respiration a number of fine moist râles were heard in the back and in the axillary regions. During the next week no change took place in the temperature, pulse, respiration, or physical signs. Some days later the temperature fell to the normal, the respirations to 36, the pulse to 135, the dulness began to disappear, and the numerous coarse and fine moist râles of resolution appeared. Resolution took place rapidly, and a week later, thirty days from the onset of the attack, the lung appeared to be in a perfectly normal condition. From that time the child gained rapidly in strength and weight and recovered completely.

The following cases illustrate the type of a central pneumonia. The showed the clinical symptoms of a lobar pneumonia, quick respiration rapid pulse, dilatation of the alæ nasi, apathy, delirium, and vomiting, b the physical signs did not appear in the lung for a number of days. Th whole course of the disease and the physical signs were so similar in bot instances that one description will suffice for both.

There were two boys, brothers, the older boy being three years and the younge sixteen months old. The older boy was attacked on November 19, and the younger one on November 20, with continuous vomiting, which lasted without much intermission until November 26. In addition to the vomiting the temperature rose in the first twenty-four hours to 40.5° C. (105° F.), and until November 26 varied from 40° to 40.5° C. (104° to 105° F.). The respirations varied from 40 to 50, and the pulse from 150 to 160. Both children soon became unconscious, were very restless, rolled their heads continuously, and showed contracted pupils. On November 26 the temperature fell to 39.4° C. (103° F.), and during the next two days varied from 39.4° to 40° C. (103° to 104° F.). On November 27 a small area of flatness with bronchial respiration was detected in the older boy over the left upper lobe in front, and on the following day in the younger boy over the left lower lobe behind. After the first few days there was slight cough in both cases, with movement of the alæ nasi. On November 30 the temperature in both children rose to 40.5° C. (105° F.), and until December 2 it varied from 40° to 40.5° C. (104° to 105° F.). On the evening of December 2 the temperature in the older boy suddenly fell from 40.5° C. (105° F.) to 35.5° C. (96° F.). The child became cold, the pulse became feeble, and the respiration could scarcely be detected. The application of a hot pack and an enema of hot brandy-and-water rapidly revived the child. The same fall of temperature occurred in the other boy on the following morning. In both children signs of resolution were detected before the temperature fell, the lungs in both cases rapidly became normal, and after a short convalescence the children recovered completely.

In some cases lobar pneumonia may attack both lungs. Again, after the disease has run its course and the temperature has fallen to normal, a fresh portion of the lung may be involved and the temperature may rise again. In rare instances in otherwise typical cases of lobar pneumonia I have been unable to detect any râles over the area of solidification throughout the whole course of the disease.

COMPLICATIONS AND SEQUELÆ.—The complications of pneumococcus lobar pneumonia are not numerous.

Pleurisy always occurs if the inflammation is superficial. It may be dry, fibrinous, sero-fibrinous, or purulent (empyema). The presence of exudation renders the diagnosis much more difficult, and aspiration is usually necessary to determine the condition.

Pericarditis may occur, and is more common in children than in adults. It is frequently associated with pericardial exudation, which is either serous or purulent. Recovery may occur even in a purulent pericarditis (Murchison).

Otitis media is not an infrequent complication in children. Often the only suggestion of its presence is a sudden rise in the temperature.

Gangrene of the lung is of very rare occurrence, but may be the râles. The voice-sounds are intensified, seem near to the ear (bronchophony), and sometimes have a nasal quality (egophony).

VARIETIES.—Certain variations in the type of pneumococcus lobar pneumonia occur, and have been designated by especial names. In *apex pneumonia* the inflammation is limited to the apex of the lung. In *massive pneumonia* the exudation fills even the bronchi, completely consolidating the lung, and giving rise to physical signs which can be differentiated from pleural exudation only by exploratory aspiration. In *migratory* or *creeping pneumonia* one lobe after another is involved. *Cerebral pneumonias* are simply those forms which are associated with severe cerebral symptoms. Some writers describe an *abortive type* of pneumococcus pneumonia in which the signs are those of the stage of congestion, and the disease runs a course of from twenty-four to forty-eight hours. I, myself, have not, however, seen in children any cases in which the evidence was sufficient to justify the diagnosis.

Central pneumonia is a type of the disease especially common in children. The clinical symptoms are present, but the physical signs are absent, as the consolidation is deep-seated. The signs may not come to the surface for many days, or resolution may take place before they appear.

Delayed Resolution.—The crisis in lobar pneumonia is not always accompanied by a resolution of the consolidation; the local signs may persist, and disappear only in the course of several weeks. In these cases of delayed resolution the temperature frequently falls by lysis.

The following case illustrates a lobar pneumonia in which resolution was delayed for thirty days.

A girl, four years old, and perfectly well and strong, was suddenly attacked with vomiting, pain in the right side, and cough accompanied, according to her mother, by a reddish-brown sputum. Physical examination on the following day revealed nothing abnormal except a few fine moist râles at the base of the right lung behind. The pulse was 170, the respirations 60, and the temperature 39.4° C. (103° F.). On the following day the temperature still remained raised, and there was dulness on percussion over the lower right lobe behind, with bronchial respiration. On the following day the dulness had extended over the whole of the right lung in front and behind. The temperature varied from 38.8° to 39.4° C. (102° to 103° F.), the pulse from 150 to 160, and the respirations from 50 to 60. These symptoms continued until the eighth day from the onset of the attack, when the temperature was found to be 38.4° C. (101.2° F.), the respirations 48, and the pulse 160. During the next nine days the temperature, pulse, and respirations remained the same, and there was no change in the physical signs of the lung, except that in addition to the dulness and bronchial respiration a number of fine moist râles were heard in the back and in the axillary regions. During the next week no change took place in the temperature, pulse, respiration, or physical signs. Some days later the temperature fell to the normal, the respirations to 36, the pulse to 135, the dulness began to disappear, and the numerous coarse and fine moist râles of resolution appeared. Resolution took place rapidly, and a week later, thirty days from the onset of the attack, the lung appeared to be in a perfectly normal condition. From that time the child gained rapidly in strength and weight and recovered completely.

secondary form of broncho-pneumonia, is a primary disease, characterized by a sudden onset and a regular temperature, which rises abruptly. This is accompanied by a corresponding rapidity of the pulse and respirations, dulness on percussion usually involving and limited to one lobe or one lung, with increased vocal fremitus and resonance, and bronchial respiration over the dull area. This is followed by a fall of temperature and by a rapid resolution. The duration is short and definite. Broncho-pneumonia, on the other hand, is usually secondary to a preceding bronchitis, occurring either alone or in the course of some other disease. It is characterized by a slow and insidious onset, except when it occurs in the course of measles; it has an irregular temperature, the rise usually not being so sudden or so high as in lobar pneumonia, and the respirations and pulse slowly increase with the temperature. There is often an absence of change in percussion, the dulness if present showing itself in small patches and commonly in both lungs. There is also often an absence of marked vocal fremitus or vocal resonance, and of bronchial respiration, except where the patches of dulness are pronounced. Moist râles of all sizes may be heard in circumscribed areas throughout both lungs. The temperature is usually of a remittent type, and this condition lasts for weeks rather than days, the duration often being prolonged. If these pictures of the two diseases are borne in mind, an error in the differential diagnosis will seldom be made. In the doubtful cases, in which the characteristic course of either disease is absent, it will usually be found that we are dealing with a case of broncho-pneumonia, which is an exceedingly variable disease, rather than with lobar pneumonia, in which some of the characteristic features of the disease are almost invariably present.

The primary forms of broncho-pneumonia, as described on page 691, are very difficult to differentiate from acute pneumococcus pneumonia, especially when the consolidation is central, and in these cases we are generally obliged to wait until the physical signs have become distinctive.

Another condition in which the diagnosis is very obscure occurs in those cases of broncho-pneumonia in which the areas of consolidation have, by confluence, involved the whole or a greater part of a lobe.

From Meningitis.—Pneumonia in children may be entirely masked by the intensity of the cerebral symptoms. In making the diagnosis between pneumonia and meningitis it is of much aid to remember that the slow intermittent pulse, slow irregular respiration, and moderate temperature of meningitis are uncommon in lobar pneumonia, in which in most cases the pulse is rapid and regular, the respirations rapid, and the temperature high. It is not uncommon, however, to find irregularities and intermissions in the rapid pulse of pneumonia. The younger the individual the more likely are the symptoms of tubercular meningitis to be replaced by those of the non-tubercular form of the disease, which may often simulate closely the symptoms of pneumonia. The convulsions which occur in pneumonia do not differ from those which occur in meningitis, or, in terminal process of a delayed resolution. In the same way an *abscess* of the lung may develop; it may perforate into the pleural cavity and produce an empyema, or into a bronchus and the pus be expectorated.

Other rare complications due to the pneumococcus are meningitis, endocarditis, and peritonitis.

Diagnosis.—The diagnosis of pneumococcus lobar pneumonia, when the typical temperature and the characteristic physical signs are present, is not difficult, but there are a number of atypical cases in which a doubt might easily arise for a number of days after the invasion of the disease. When a specimen of the sputum can be obtained, the character of the infection can often be determined at once, even when the physical signs are not distinctive, by a bacteriological examination in reference to the presence of the pneumococcus in large numbers, alone or associated with other organisms, within the leucocytes.

From Pleural Exudation.—An early diagnosis from a pleural exudation is at times impossible, and is always quite difficult in the early stages before the characteristic signs of consolidation have been established. In both diseases dulness or flatness over a limited area, and bronchial respiration without any especial difference in the tactile fremitus and vocal resonance, and without evidence of a friction-rub or of râles, may make the two diseases simulate each other closely and compel us to wait for further developments before determining the differential diagnosis. This similarity in the signs of fluid and consolidation is especially characteristic in children. In typical cases of pleural exudation the absence or marked diminution of tactile fremitus and vocal resonance, the shifting character of the area of dulness on change of position, the diminished intensity of the respiratory sounds, bulging of the intercostal spaces, and displacement of the heart, and of the liver in exudations on the right side, are signs which render definite the diagnosis from a consolidated lung. In atypical cases, however, the signs may so closely resemble those of pneumonia that the diagnosis can only be made by exploratory puncture.

From Tuberculosis of the Lung.—In tubercular disease of the lung the differential diagnosis is usually not difficult, except in young infants, in whom the tubercular process with its corresponding symptoms may in rare cases simulate lobar pneumonia. The onset of the symptoms, the course of the disease, and the examination of the sputum, will, in nearly all uncomplicated cases, make the diagnosis clear.

From Broncho-Pneumonia.—The disease from which a differential diagnosis should especially be made is broncho-pneumonia. Lobar pneumonia and secondary broncho-pneumonia are so distinct, however, in their previous history, initial stage, course, and duration that, if care be taken to note closely all these stages of the two diseases and to arrive at a diagnosis from the evidence given by all the stages and not by any one stage, the diagnosis can, except in the very early days of the disease, usually be determined. Lobar pneumonia, in contradistinction to the serious symptoms should arise. These remedies should be the external application of heat by means of the hot pack, and the administration of brandy by the mouth if the child can swallow, otherwise by rectal injection. I have known of a case where a child died in the collapse following the crisis of a lobar pneumonia. It is often not necessary to use any remedy whatever, and it is safer to wait until there are indications for the use of drugs. There is no remedy which will shorten the course of the disease. Cardiac weakness, hyperpyrexia, pain, and toxæmia are the four indications which require special symptomatic treatment.

Cardiac Weakness.—The condition of the circulation should be carefully watched. When the disease occurs in very young infants it is safer to administer stimulants from the beginning. Brandy, or whiskey, and strychnine are the most serviceable drugs for a cardiac stimulant. The brandy should be adapted to the age of the child as given on page 456, and the strychnine as stated on page 470. The limits of administration will depend upon the way in which the heart responds to the stimulant. Inhalation of oxygen often gives great relief when there is cyanosis and marked dyspnœa. Aromatic spirits of ammonia may also be used to supplement the alcoholic stimulation.

Hyperpyrexia.—Although at times a high temperature does not necessarily indicate danger, since a temperature of from 40° to 40.5° C. (104° to 105° F.) is part of the regular course of the disease, yet if the temperature rises above this point it is well to reduce it by means of bathing and to give alcoholic stimulants. When the child does not react well to the bath, phenacetine in doses of 0.06 to 0.12 gramme (1 to 2 grains) will often keep the temperature down for several hours. It should be guarded with stimulants. It must, however, be understood that it is not the height of the temperature so much as the intensity of the nervous symptoms which determines the indication for antipyretic treatment.

Pain.—Pain is often a distressing symptom, and may at times require small doses of paregoric, adapted to the age of the child. From one to three months, 0.06 to 0.12 c.c. (1 to 2 minims) may be given; at one year, 0.3 to 0.6 c.c. (5 to 10 minims); and at five years, 1.8 to 2.4 c.c. (30 to 40 minims). The application of ice-poultices is indicated in these cases, but only for short periods.

Toxæmia.—Although extensive investigations have been made in regard to the serum therapy of pneumococcus lobar pneumonia, the efficiency and expediency of the treatment is still in doubt. The only means we possess of combating the toxins is by reduction of the fever and by general supportive treatment.

Convalescence.—During convalescence the child should be carefully protected from atmospheric changes, cold, and dampness for some months, and its food and habits should be carefully regulated.

Lobar pneumonia may occur in the earliest days of life. I have met with a case which on the third day of its life developed a lobar pneufact, in any other acute disease. A careful physical examination should be made at every visit, once or twice a day if possible, as in this way the masked symptoms of a pneumonia may at times be detected where they would be overlooked if only an occasional examination were made. After the first four or five days, as a rule, the differential diagnosis between cerebral disease and pneumonia is not difficult. Lumbar puncture is of much value in the diagnosis of meningitis in these cases.

From Influenza.—The presence of a well-marked leucocytosis is of much service in excluding influenza, but the possibility of an influenza pneumonia must be borne in mind. In the latter case, if leucocytosis is present at all, it is rarely high, and the examination of the sputum, the course of the disease, and the character of the physical signs generally make the diagnosis clear.

From Malaria.—The onset of malaria may closely resemble that of lobar pneumonia, but its subsequent course is essentially different. A chill with a sharp rise in the temperature, associated with a marked leucocytosis, is rarely, if ever, due to malaria.

PROGNOSIS.—The prognosis of pneumococcus lobar pneumonia is very favorable. In young infants, or in those who are weak and debilitated, it is often fatal, but in comparison with broncho-pneumonia the percentage of recovery is very high. When the temperature rises to 41.1° C. (106° F.) the prognosis is usually grave. The convulsions which occur in the initial stage of the disease in infants are commonly not of grave import. Occurring late in the disease they make the prognosis very unfavorable. When delirium occurs, although it may be severe, it does not render the prognosis especially bad. The fulminant type of the disease which sometimes occurs is a very fatal form.

TREATMENT.—As lobar pneumonia is a self-limited disease of short duration, the children are not so apt to die of exhaustion, and, as a rule, only an expectant treatment is called for.

Nursing and General Hygiene.—The child should be placed in a room of an equable temperature of 20° or 21.1° C. (68° or 70° F.), and should be given milk every two hours. There is no necessity for making any external applications to the chest. The use of poultices is to be deprecated, and in my experience is usually without benefit except in certain instances for the relief of pain. The nursing is of especial importance, and close watchfulness, especially at the time when the crisis is expected to take place. At this time the temperature in infants and young children may fall with such rapidity to several degrees below the normal point that collapse often takes place, the skin is cold and moist, and sometimes the child becomes unconscious. Under these circumstances the pulse is feeble and intermittent, and in some cases death may occur unless active measures are taken for establishing reaction. The nurse should therefore be warned as the time of the expected crisis approaches to watch the child both night and day, and to have remedies ready to be used in case serious symptoms should arise. These remedies should be the external application of heat by means of the hot pack, and the administration of brandy by the mouth if the child can swallow, otherwise by rectal injection. I have known of a case where a child died in the collapse following the crisis of a lobar pneumonia. It is often not necessary to use any remedy whatever, and it is safer to wait until there are indications for the use of drugs. There is no remedy which will shorten the course of the disease. Cardiac weakness, hyperpyrexia, pain, and toxæmia are the four indications which require special symptomatic treatment.

Cardiac Weakness.—The condition of the circulation should be carefully watched. When the disease occurs in very young infants it is safer to administer stimulants from the beginning. Brandy, or whiskey, and strychnine are the most serviceable drugs for a cardiac stimulant. The brandy should be adapted to the age of the child as given on page 456, and the strychnine as stated on page 470. The limits of administration will depend upon the way in which the heart responds to the stimulant. Inhalation of oxygen often gives great relief when there is cyanosis and marked dyspnœa. Aromatic spirits of ammonia may also be used to supplement the alcoholic stimulation.

Hyperpyrexia.—Although at times a high temperature does not necessarily indicate danger, since a temperature of from 40° to 40.5° C. (104° to 105° F.) is part of the regular course of the disease, yet if the temperature rises above this point it is well to reduce it by means of bathing and to give alcoholic stimulants. When the child does not react well to the bath, phenacetine in doses of 0.06 to 0.12 gramme (1 to 2 grains) will often keep the temperature down for several hours. It should be guarded with stimulants. It must, however, be understood that it is not the height of the temperature so much as the intensity of the nervous symptoms which determines the indication for antipyretic treatment.

Pain.—Pain is often a distressing symptom, and may at times require small doses of paregoric, adapted to the age of the child. From one to three months, 0.06 to 0.12 c.c. (1 to 2 minims) may be given; at one year, 0.3 to 0.6 c.c. (5 to 10 minims); and at five years, 1.8 to 2.4 c.c. (30 to 40 minims). The application of ice-poultices is indicated in these cases, but only for short periods.

Toxæmia.—Although extensive investigations have been made in regard to the serum therapy of pneumococcus lobar pneumonia, the efficiency and expediency of the treatment is still in doubt. The only means we possess of combating the toxins is by reduction of the fever and by general supportive treatment.

Convalescence.—During convalescence the child should be carefully protected from atmospheric changes, cold, and dampness for some months, and its food and habits should be carefully regulated.

Lobar pneumonia may occur in the earliest days of life. I have met with a case which on the third day of its life developed a lobar pneumonia and died in twenty-four hours. The autopsy showed the characteristic hepatization of lobar pneumonia.

The following case illustrates lobar pneumonia :

A boy, eight years old, entered the hospital on the fourth day of an attack of lobar pneumonia. The attack began with vomiting and cough, but no pain, expectoration, or chill. An examination showed the right lung to be normal. On the left side of the chest an area corresponding to the lower lobe in the back was found to show dulness on percussion, bronchial respiration, and many fine moist râles. Nothing else abnormal was detected. The child was very restless, but on the following day, the fifth from the onset of the disease, the temperature fell by crisis to the normal point. Chart 28 shows the typical temperature, pulse, and respiration of a case of lobar pneumonia.

After the crisis the child improved rapidly, and the physical signs disappeared in ten days.

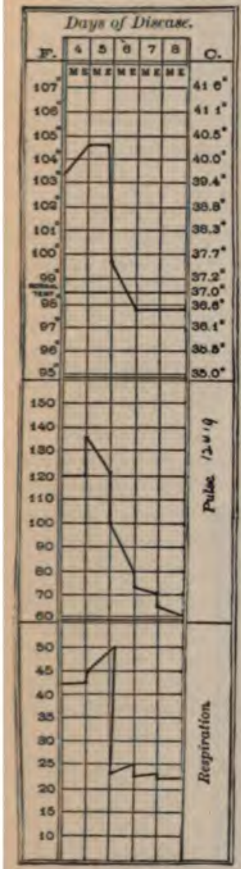
A girl, two and a half years old, entered the hospital on the third day of an attack of lobar pneumonia. Chart 29 shows one of the variations in the crisis which is quite frequently met with in young children. The temperature in this case reached the normal on the tenth day.

In this case the consolidated portion of the lung was the left lower lobe. The resolution was rapid and convalescence normal.

A girl (Fig. 143), eight years old, had pertussis when she was fourteen months old, scarlet fever when she was five years old, and measles when she was six years old. Five days before entrance she lost her appetite, was very feverish, and was attacked with acute pain referred to the left side of the epigastrium and the lower part of the left axillary region. This was accompanied with a hacking paroxysmal cough, with no expectoration. She became delirious, vomited occasionally, and was very weak. Her tongue was heavily coated, the alæ nasi moved with respiration, her face was deeply flushed, and the dyspnœa was so severe that she had to be propped up on pillows.

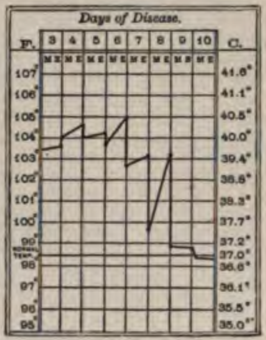
Her respirations were 45, difficult and painful, her pulse 120, and her tempera-

CHART 28.



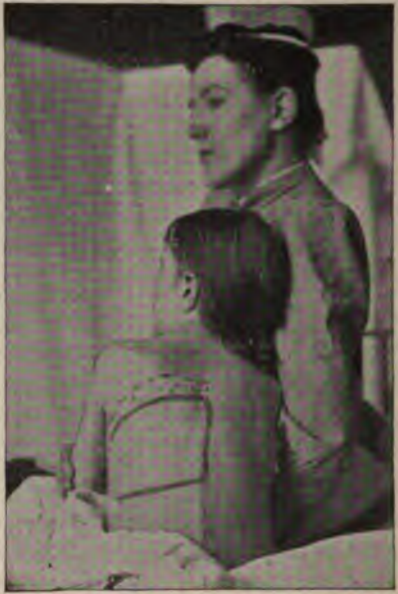
Lobar pneumonia. Male, 8 years old. Crisis on fifth day of disease.

CHART 29.



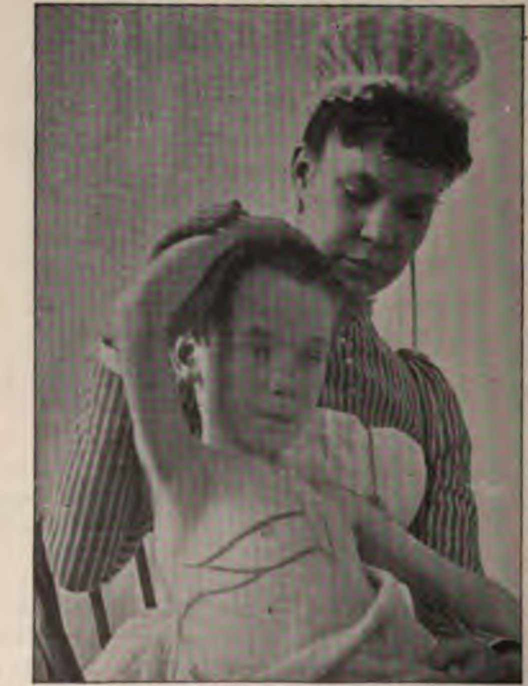
Lobar pneumonia. Irregular crisis on eighth day. Female, 2½ years old.

Fig. 143.



Lobar pneumonia. Female, 8 years old. The part of the lung involved by the pneumonic process is shown by black lines, and the area of diminished resonance and the fine râles are marked by black spots.

Fig. 144.



Lobar pneumonia. Three invasions. Male, 6 years old.

39.5° C. (103.2° F.). A physical examination detected nothing abnormal in the of the chest or in the right back. There was flatness in the left back, beginning fifth rib and extending to the base of the lung and into the axillary region. this area there was increased vocal fremitus and bronchial respiration, and a few râles. Just above the upper border of the area of flatness there were diminished ance and a number of fine râles. Although the examination was made on the lay of the disease, the physical signs showed that resolution had begun and that isis might be expected at any time.

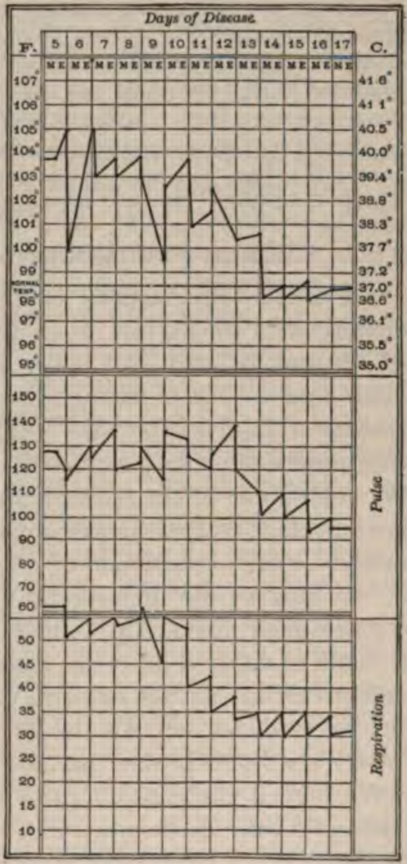
n the following day the temperature fell to 37.7° C. (100° F.) in the morning, ose again in the evening to 39.1° C. (102.5° F.). On the following day, the th day from the beginning of the attack, emperature fell to 37.2° C. (99° F.), hen varied from 37.7° C. (100° F.) to C. (99° F.) until the eleventh day, it became normal. The pulse and ations declined synchronously with the erature.

his case illustrates the fact that the cal signs of resolution may sometimes r before the temperature falls and the comes; also that at the time of the the temperature may fall, then rise for from twelve to twenty-four hours, hen fall to the normal. The child re-ed completely.

nother child, a boy (Fig. 144), six old, was taken sick four days before ng the hospital.

n entering the hospital his pulse was his respirations 60, and his temperature C. (103.8° F.) A physical examina-howed that there was flatness over the upper lobe of the right lung; over rea there were bronchial respiration, sed vocal resonance, and an occa-l high-pitched râle. The left lung was al. The lower border of the dulness ced by the consolidated upper lobe has marked by a black line extending from ternum just above the right mamma round into the axillary region. On orning of the sixth day from the be-ng of the attack the temperature fell .7° C. (100° F.), but rose again in the ng to 40.5° C. (105° F.) and a physical ination then showed that the middle f the right lung was involved in front, as indicated by the second black line the one just described. The temperature during the next two days remained en 39.4° and 40° C. (103° and 105° F.), but on the following day, the ninth from nset of the disease, the temperature suddenly fell to 37.6° C. (99.7° F.) in the ng, but rose the next morning to 39.3° C. (102.8° F.), and in the evening was C. (103.8° F.). A physical examination then showed that the whole of the lobe was involved, as indicated by the third black line. On the following day

CHART 30.



Lobar pneumonia of the migratory type. Male 6 years old.

the upper lobe began to show evidence of resolution, and the temperature fell to 38.3° C. (101° F.). Two days later the temperature began to fall by lysis, the physical signs of the upper and middle lobes entirely disappeared, and the temperature reached the normal point on the fourteenth day from the time of the onset. On the seventeenth day from the time of the onset the lower lobe was also found to be in a normal condition, and from that time convalescence was uninterrupted.

Chart 30 shows the temperature, pulse, and respirations in this case from the fifth to the seventeenth day of the disease.

In some rare cases the infection in lobar pneumonia is so overwhelming that a rapidly fatal issue may occur.

I have seen a little girl, nineteen months old, who had been having so mild an attack of diarrhœa that she was playing about out of doors, suddenly attacked in the afternoon with convulsions and a temperature of 40.5° C. (105° F.). The convulsions continued during the night, and she soon became comatose. On the following day the temperature still remained at 40.5° C. (105° F.), the respirations were much accelerated, and the pulse was about 120. An area of absolute dulness over the left lower lobe behind, with bronchial respiration and increased vocal resonance and fremitus, rapidly developed. The child did not respond to treatment, and died in the evening.

LOBAR PNEUMONIA DUE TO OTHER ORGANISMS.

It occasionally happens in a very small percentage of cases, as has already been stated, that in an acute infection of the lung an entire lobe, or the greater part of a lobe, may become consolidated in conditions other than that of a pneumococcus lobar pneumonia. This fact and the custom of applying the term "lobar pneumonia" to inflammations of the lung produced by the pneumococcus have led to more or less confusion in the description of the etiology and classification of the various forms of pneumonia. Reference has already been made on page 434 to the rare cases of total consolidation of a lobe occurring as a result of an infection of the lung by the diplococcus intracellularis secondary to an epidemic cerebrospinal meningitis. Other cases occur in which the pneumococcus is found in a mixed infection, associated with some form of pus-producing organism, such as the staphylococcus pyogenes aureus and the streptococcus pyogenes, or with the bacillus of influenza, the bacillus of Friedländer, the Klebs-Loeffler bacillus, and rarely the typhoid bacillus. Moreover, there is the class of cases in which the etiology and pathology are those of a pure broncho-pneumonia, in the course of which all the lobules of the affected lung have become confluent, giving rise to the gross appearances and physical signs of a lobar consolidation. Whether a lobar pneumonia with the gross and microscopic appearances of a typical pneumoccoccus lobar pneumonia can ever be due to a single organism other than the pneumococcus is a question which the present state of our knowledge of the etiology and pathology of the disease does not warrant us in answering definitely.

Lobar pneumonias which are caused in the manner just outlined often differ in their clinical symptoms and course from those of a pneumococcus lobar pneumonia. They are much more apt to be subacute at their onset, secondary to some other infection, and to run a more irregular course, resembling closely that of a broncho-pneumonia. The bacteriological examination of the sputum is of the greatest aid in these cases, and may often, in connection with the clinical symptoms and physical signs in the early stages of the disease, enable us to make a differential diagnosis.

Treatment.—The treatment is symptomatic, and does not differ from that of pneumococcus lobar pneumonia or of broncho-pneumonia.

ACUTE BRONCHO-PNEUMONIA.

Acute broncho- or lobular pneumonia is an infection of the lung characterized by an inflammation of the walls of the terminal bronchi and of the neighboring alveoli which make up a pulmonary lobule, and is so called in contradistinction to lobar pneumonia.

Etiology.—All forms of broncho-pneumonia are produced by pathogenic organisms. The disease may be primary or secondary, acute or chronic. It may occur at any age, but is the most common and fatal form of inflammation of the lung during the first five years of life, and is much more fatal than pneumococcus lobar pneumonia during this period.

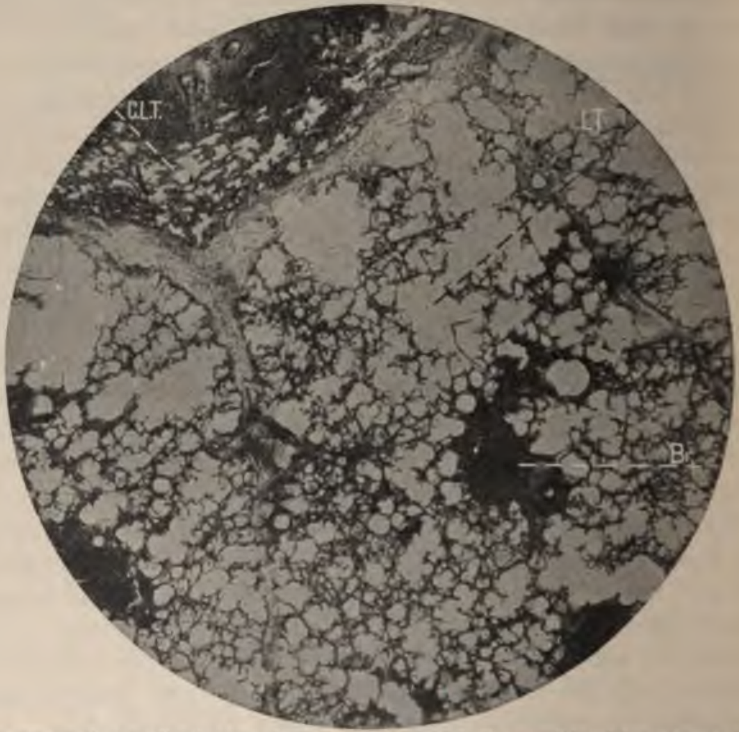
The *primary forms* may attack children in good health, and usually under two years. It is probable that the pneumococcus is usually the infecting organism in these cases, and that the lobular rather than the lobar type of the disease is determined by the greater susceptibility of the lung from its embryonic type to the kind of inflammatory lesions which characterize a broncho-pneumonia. The bacillus of Friedländer and the bacillus of influenza produce this especial type of pneumonia.

The *secondary forms* are much more frequent than the primary forms, and are very apt to be the result of a mixed infection of streptococci, staphylococci, and pneumococci. They are especially fatal in infants and young children. They occur in connection with the infectious fevers, and with the group called the *aspiration* or *deglutition pneumonias*, which sometimes follow vomiting of food, especially after ether, hæmoptysis, bronchiectasis, and empyema. The relative frequency in 443 cases, according to Holt, is as follows: primary, without previous bronchitis, 164; secondary to measles, 89; whooping-cough, 66; diphtheria, 47; bronchitis, 41; acute ileo-colitis, 19; scarlet fever, 7; influenza, 6; varicella, 2; and erysipelas, 2.

Children who are weak or debilitated by previous diseases show a predisposition to broncho-pneumonia, which frequently arises in the course of tuberculosis, chronic gastro-enteric diseases, and rhachitis. Those seasons of the year which are marked by cold, moisture, and variations of temperature especially predispose to the development of broncho-pneumonia. All these conditions, however, in all probability merely prepare the way for the entrance of certain germs which produce the disease. The origin of broncho-pneumonia from intestinal infection must also be considered.

PATHOLOGY.—In broncho-pneumonia the inflammatory process affects the walls of the smaller and terminal bronchi, which become thickened and markedly infiltrated with cells. The inflammatory process then extends through the walls of the bronchi to the surrounding air-vesicles as well as to the terminal ones. In this way centres of consolidation are formed in different parts of the lung. The course of the inflammation varies in its rapidity, at times attacking only a small portion of the lung, and again being more diffuse in its onset and gradually invading large areas. The lesions are irregular in their distribution and usually occur in both lungs. They are at times so extensive as to involve a whole lobe, but, as has been stated by Northrup, whatever the extent of hepatization, whatever the time occupied in its course, and whatever the post-mortem appearances, the essential lesion is an inflammation of the walls of the terminal bronchi and of the adjacent alveoli.

FIG. 145.



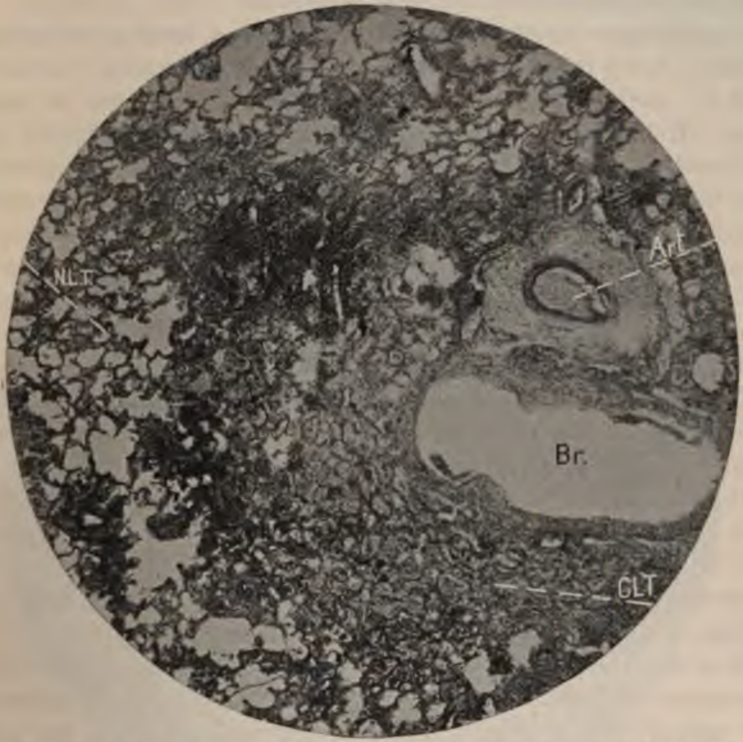
Broncho-pneumonia complicating measles. Early stage. C. L. T., consolidated lung-tissue; Br., bronchiole; L. T., emphysematous lung-tissue.

Fig. 145 represents the section of a lung made by Northrup, and taken from an infant sixteen months old, in whom the broncho-pneumonia was a complication of measles. It shows the early pathological lesions of broncho-pneumonia.

In one of the lobules there are two bronchioles (Br.) with infiltrated walls and pus within them. They are also filled with exudation, and the lumen of each is almost entirely occluded. A portion of a neighboring lobule is consolidated (C. L. T.). A considerable portion of the lung tissue (L. T.) in the section is normal or emphysematous.

Fig. 146 represents another section taken from the lung of the same child, but showing the tissue relatively less affected. Many of the consolidated alveoli contain free blood-cells. The bronchial wall (Br.) is infiltrated and almost entirely denuded of its lining membrane. These smaller bronchi are surrounded by zones of intense congestion and infiltration. When the inflammation is intense and is accompanied by abundant secretion these bronchi frequently become dilated. This dilatation is associated with a weakened condition of the bronchial walls and with an abundant secretion. These dilatations probably, according to the observations of Northrup, wholly disappear on the recovery of the patient.

FIG. 146.



Broncho-pneumonia secondary to diphtheria. Br., bronchus; C. L. T., consolidated lung-tissue; N. L. T., lung-tissue nearly normal; Art., artery.

As has already been described in the pathology of bronchitis, the bronchial lymph-nodes are always enlarged in broncho-pneumonia, and there may be fibrin on the pulmonary pleura. In the zones of peribronchitic pneumonia the walls of the air-vesicles are thickened or swollen, either with or without some cellular infiltration, and the cavities of the air-vesicles are filled with epithelial cells and pus-cells, with fibrin and red blood-corpuscles in varying proportion and amount. Fibrin when present is only in small quantities, and often is altogether absent. The capillaries in the walls of the vesicles are congested and prominent. The portions of lung which are not hepatized are congested and œdematous. The cavities of the air-vesicles are diminished by the enlarged capillaries and the swollen vesicular epithelium.

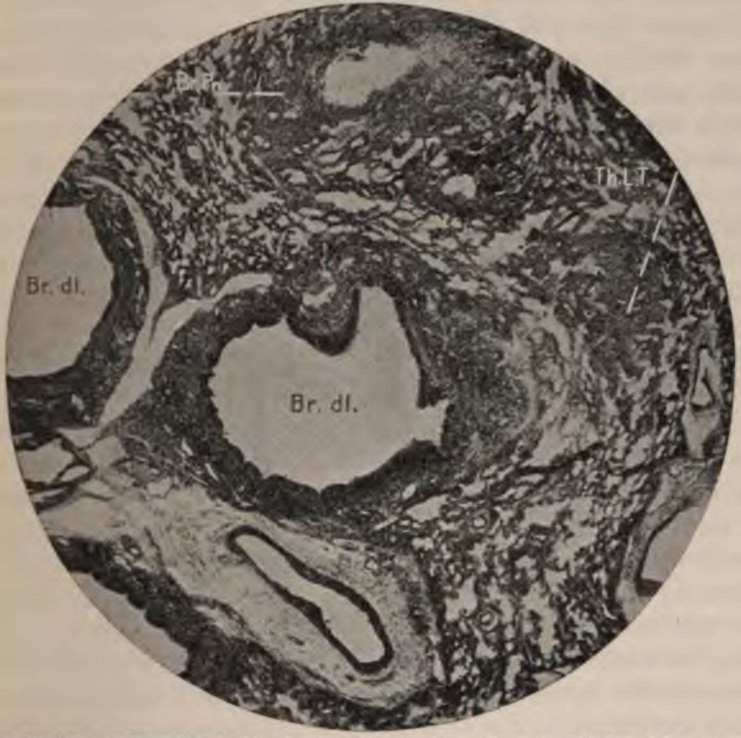
In addition to the other lesions just described, areas of atelectasis are frequently found in broncho-pneumonia. This atelectasis is usually produced by mechanical causes, such as obstruction by pus or tenacious mucus. It may also arise as a result of enfeebled respiratory power. The blood-vessels become dilated, the walls of the alveoli partially collapse, the residual air is absorbed, and an exudation of serum with proliferative cells and leucocytes takes its place. The atelectasis is commonly symmetrical, affecting the posterior margin of both lower lobes of the lung, but it may also appear in irregular scattered areas in the posterior portions of the upper lobes. It may occur either during the acute stage of the inflammation or later when the pneumonia has become chronic.

There are no distinct stages in the pathology of broncho-pneumonia which correspond to those of lobar pneumonia. Broncho-pneumonia develops by the irregular invasion of successive portions of the lungs, and the process resolves in like manner. The different consolidated areas in the same lung may often show all the stages. The mottled appearance which is so often noticed macroscopically in these lungs may be caused by the presence of lobules of gray and red hepatization lying side by side. Of these inflammatory products the fibrin disintegrates quickly, and is therefore absorbed more rapidly than the cellular elements, which do not disintegrate so readily. In lobar pneumonia, therefore, absorption takes place sooner than it does in broncho-pneumonia, in which the products of inflammation are mostly cellular and resolution and absorption are naturally slow.

Instead of the gradual disappearance of the various pathological lesions the pneumonia may persist. This persistent form of the disease may follow a single attack of acute broncho-pneumonia, or there may be several acute attacks before the chronic condition becomes evident, and the course of the disease may thus vary in different cases. When this persistent broncho-pneumonia occurs, the proliferative cells take part in the formation of new connective tissue, and in this way persistent thickening is caused. The alveolar walls of certain portions may become similarly thickened. The walls of the bronchi and their surrounding tissue are especially subject to a persistent thickening and a formation of new connective tissue constituting chronic broncho-pneumonia and peribronchitis. The bronchi already dilated become still more enlarged by the contraction of the cicatricial tissue surrounding them. The uneven contraction of this ew tissue, together with the pressure within the tubes facilitated by a eakened condition of the walls, allows of saccular as well as of fusiform ilatation of the bronchi. The epithelial cells of the dilated bronchi proferate, and, falling from the bronchial walls, mix with the bronchial ecretion. The remaining epithelium is swollen and loose. The lesions f chronic broncho-pneumonia are frequently associated with tuberculosis f the bronchial nodes and with other tubercular lesions.

In connection with the pathological lesions occurring in chronic roncho-pneumonia a condition called *fibroid phthisis* has in very rare ases been noticed in children. The lesions which represent fibroid hthisis are manifested by the presence of connective tissue in the lung, ith a corresponding destruction of the true parenchyma. These hanges are usually unilateral, and should not be considered as representing a disease, since they merely occur in the course of various chronic ulmonary affections, among which are tuberculosis and chronic bronchoneumonia.

FIG. 147.



Chronic broncho-pneumonia. Br. dl., dilated bronchus; Th. L. T., thickened lung-tissue Br. Pn., broncho-pneumonia.

Fig. 147 represents a section, also made by Dr. Northrup, taken from lung with chronic broncho-pneumonia in which the process had adanced still further than in the other.

In the middle of the specimen is a dilated bronchus with a section of a blood-vessel just below it. There is considerable connective-tissue formation about both. In this case the process of a peribronchitic pneumonia has gone further than in the other specimen (Fig. 145), and there is, in addition to the dilated bronchi with the surrounding cellular infiltration, a tendency to the formation of connective tissue in the interlobular septa. This is the form of chronic broncho-pneumonia which is sometimes called interstitial pneumonia, and is usually characterized by a long course and delayed recovery.

A frequent lesion which occurs in the course of broncho-pneumonia is *emphysema*. It is usually vesicular and situated in the anterior portion of the upper lobes. It is due to the diminished amount of air-capacity, together with the violent introduction of air into the chest caused by dyspnœa and coughing. This distention of the air-vesicles is supposed usually to disappear with the subsidence of the lesion which is causing the emphysema. Emphysema, both of the vesicular and of the interstitial variety, most commonly occurs in the pneumonia which follows pertussis.

The interstitial variety may exist in the form of superficial sacs, formed by the rupture of air-vesicles beneath, which lift the pleura, or it may extend between the lobules in V-shaped tracts from the anterior edge of the upper lobe even to the root of the lung.

SYMPTOMS.—The symptoms of broncho-pneumonia vary greatly in their onset and course, owing to the many different lesions which commonly occur in the disease and which by their greater or less severity make its course exceedingly irregular. In so many instances the broncho-pneumonia is secondary to some other disease that the symptoms are necessarily modified by those of the initial affection. Thus, when broncho-pneumonia arises in the course of diphtheria, the symptoms are often obscured by the severity of the general symptoms of the diphtheria. When broncho-pneumonia is secondary to measles and to pertussis, although at times its onset is difficult to detect, yet, as a rule, the rapid respirations, the marked and continuous rise of temperature, and the evident exacerbation in the severity of the pulmonary symptoms, usually permit a diagnosis to be made even before the physical signs have become prominent. Its onset, however, in measles is, as a rule, rapid, while in pertussis it is slow and insidious.

The group of symptoms which characterizes a broncho-pneumonia arising during the course of bronchitis is somewhat more definite. In place of the moderate temperature and the absence of signs of serious disease which are usually met with in the course of an ordinary bronchitis, when broncho-pneumonia supervenes the temperature rises, the pulse and respirations are increased in frequency, the alæ nasi dilate, there is more or less cyanosis, the cough becomes more frequent and painful, and the general aspect of the patient is that of one suffering from an affection of a severe type.

Onset.—The onset of broncho-pneumonia varies according as the disease is primary or secondary. In the *primary form* the symptoms may be mistaken for a lobar pneumonia. They begin abruptly with a chill or convulsion, with a rapid rise in the temperature, which is more constant than in the secondary forms. The signs are more local in character and the disease may be masked by the intensity of the cerebral symptoms.

The *secondary form* begins as an inflammation of the bronchioles, and is not to be distinguished from a *capillary bronchitis*, which is essentially the same disease. The onset is rarely sudden, and there is no chill as a rule. It is frequently associated with one of the infectious fevers, occurring during the course of the disease or in early convalescence, or as a result of an inhalation of foreign bodies. The child appears fretful and restless. The temperature rises and is very variable. There is cough, dyspnœa, and rapid respiration. The physical signs are often limited to scattered localized areas of fine and medium moist râles, and sometimes sonorous and sibilant râles; dulness may or may not be present in this early stage.

Temperature.—The temperature in broncho-pneumonia varies greatly, according to the extent and severity of the lesions. Corresponding to the intensity of the pneumonic onset, or to the especial disease which it complicates, the temperature rises rapidly or slowly and insidiously. The most common course in mild cases with gradual onset and terminating in recovery is for the temperature to rise gradually to 39.4° or 40° C. (103° or 104° F.), then to have a morning remission of three or four degrees for a number of days, and finally to fall irregularly by lysis. A crisis is very rare in broncho-pneumonia, but sometimes occurs in the primary form of the disease. Although the remissions in the temperature during the active stage of the disease are often quite marked, yet, as a rule, the temperature does not at this time fall to the normal. This is of service in differentiating certain cases of broncho-pneumonia, as well as of lobar pneumonia, from malaria. In the more severe and unfavorable cases the temperature becomes more and more elevated, even reaching as high as 41.6° C. (107° F.). Occasionally the temperature is reversed, the highest point being reached in the morning. This is rare, and is of no especial significance. When the temperature instead of remitting remains high and steadily rises, the disease, as a rule, soon terminates fatally. Instead of the continued high temperature which occurs so often in fatal cases, a low temperature of only a few degrees above normal is sometimes met with, usually when the vitality is low and the power of reaction slight. The duration of the heightened temperature is very variable, and may last for a number of days or for weeks without the necessary result of the grave lesions of a more chronic process.

Pulse and Respiration.—The pulse and respiration, although increased in frequency, vary according to the severity of the disease and also according to the degree of nervous excitement. This latter is a very important element to be considered in determining the gravity of their rate. The pulse is at times very rapid, 160 to 180, and even higher; it usually varies from 130 to 150 or 160; although regular and full at first, it becomes weak and sometimes irregular as the disease progresses, and is very apt to remain rapid even after the temperature has declined and convalescence has been established. The respirations may be quickened by an unusually high temperature, but depend mostly on the extent of the involvement of the alveoli. They vary from 50 to 80, but they may be even higher, and are accompanied by dilatation of the alæ nasi.

The respiration often shows a pause after inspiration instead of after expiration, as occurs in normal respiration, and is usually accompanied by an expiratory moan. This sign, however, is not characteristic of broncho-pneumonia, as it may occur in lobar pneumonia and in various affections in which the circulation is interfered with and respiration is painful. In like manner the dilatation of the alæ nasi may occur in any disease accompanied by a heightened temperature and nervous excitement. Temporary exacerbations and changes in the rhythm of respiration are quite common in broncho-pneumonia, and in some cases a Cheyne-Stokes type of respiration has been noticed. This sign is usually one of grave import. Recession of the epigastrium and of the intercostal spaces commonly occurs in broncho-pneumonia, and varies according to the severity of the pulmonary lesions. In infants painful respiration is shown by a frown rather than by crying, while in young children it is shown by their whimpering and suppressed cries.

Cough.—A frequent, short, hacking, and painful cough is a constant symptom from the beginning of the disease, and even after resolution has taken place this may continue for a long period. Infants and young children, even up to the age of seven or eight years, have often not learned to expectorate, so that we cannot, as in adults, judge of the character of the sputum.

Gastro-Enteric Symptoms.—Vomiting is at times met with, and diarrhœa is not uncommon. In certain cases disturbance of the gastro-enteric tract is present from the very beginning, and the intestinal disease is apparently as prominent a feature of the attack as the pulmonary symptoms. As the attack progresses the child loses much in weight, the face often looks pinched, and at times during the height of the disease there is a certain amount of delirium, which in combination with other grave symptoms, such as uncontrollable diarrhœa and a depressed temperature, is a serious symptom.

Cyanosis and Dyspnœa.—A symptom which occurs quite commonly in broncho-pneumonia is cyanosis. This may not only arise from the interference with the oxygenation of the blood from the lesions involving the air-vesicles, but may also be produced by a temporary atelectasis of certain portions of the lungs. The cyanosis is often accompanied by attacks of dyspnœa. When these symptoms result from atelectasis, the temperature, as a rule, does not rise, but may even be somewhat reduced, and areas of dulness may be detected on percussion. During these paroxysms the skin is often cold and moist. When the cause of the atelectasis, whether it be obstruction by plugs of mucus or pus or temporary exhaustion of the contractile powers of certain portions of the lungs, has been removed, the cyanosis and dyspnœa pass away and the general symptoms improve. These symptoms may arise at various periods during the course of broncho-pneumonia, and unless the atelectasis passes off within a few days a fatal issue is very apt to result.

In cases which recover, resolution takes place slowly and the lung gradually returns to the normal condition. Great weakness and prostration often last for a long time. Relapses are quite common.

Blood.—A leucocytosis is generally present if the broncho-pneumonia is due to the pneumococcus or to mixed infections. When the process is excited by the bacillus of influenza or the bacillus of Friedländer alone, the white blood count is either normal or only slightly elevated.

Physical Signs.—The physical signs of broncho-pneumonia are almost entirely those of the accompanying bronchitis, but in typical cases they correspond to the various pathological lesions which have been described. According as larger or smaller areas of the lung are involved, corresponding areas of dulness on percussion may be found, provided these areas are sufficiently extensive not to be masked by other resonant portions of the lungs. They can, as a rule, be detected best by very light percussion. These areas of dulness are usually bilateral and of somewhat varied extent, although, as has already been stated, an entire lobe may in rare instances be sufficiently involved by the broncho-pneumonic process to produce very extensive areas of dulness. Over the area of dulness bronchial respiration, and in some cases increased vocal resonance and fremitus, may be found. On auscultation moist râles of all grades may be heard all over the lungs, or, as is more usual, in circumscribed areas.

Well-marked physical signs, especially dulness on percussion, are sometimes found at the bases of both lungs behind, and also between the scapulæ and the vertebral column. The earliest changes, however, in percussion and auscultation are sometimes first detected in the highest part of the axilla. These signs of consolidation are rarely found in the early days of the disease, as at this time signs of bronchitis are usually all that can be detected.

The absence of demonstrable areas of dulness is, however, so very common that it is almost characteristic. This may be due to the fact that the consolidation is deep-seated or in such small areas and so interspersed with uninvolved lung-tissue that although superficial in distribution it causes no perceptible change in the percussion-note. The physical signs are markedly modified when atelectasis or emphysema is present.

TERMINATIONS AND COMPLICATIONS.—*Resolution.*—If the course of the disease is favorable, the pathological process in the areas of consolidation terminates in resolution, which is often more rapid in the individual areas than in lobar pneumonia, but may be delayed for several days or weeks as in the latter affection.

Suppuration.—Resolution by suppuration is rare in the more common forms of primary and secondary broncho-pneumonias, and is limited almost entirely to cases of inhalation or deglutition broncho-pneumonias, in which it is very frequent. Under the same conditions gangrene of the lung may develop.

Chronic Fibroid Changes.—These will be described separately on page 705.

Tuberculosis.—Cases of broncho-pneumonia with delayed resolution show a special susceptibility to infection by the tubercle bacillus. This complication will be found more fully described on page 394.

DIAGNOSIS.—The diagnosis of broncho-pneumonia should first be made from the bronchitis which ordinarily accompanies it. This has already been sufficiently referred to in speaking of the diagnosis of bronchitis.

The differential diagnosis between the non-tubercular and the tubercular forms of broncho-pneumonia is important, but can rarely be made in the early stages of the disease, as the lesions are the same, and a bacteriological examination of the sputum in these cases can seldom be obtained.

The disease which should be especially considered in making the diagnosis of broncho-pneumonia is lobar pneumonia. The differential diagnosis has been given in connection with pneumococcus lobar pneumonia on page 683.

PROGNOSIS.—Age is a very important factor in the prognosis of broncho-pneumonia. As shown by Morrill a large majority of the fatal cases occurs in the first two years of life. Broncho-pneumonia causes more deaths in children than are due directly to the fevers, and is the most serious complication of the contagious diseases. The prognosis varies according to the disease in the course of which it arises. It is most grave when it occurs in pertussis, especially in infants, and the younger the child the more fatal the disease. Next to pertussis, the gravity of the prognosis is greatest in measles and in diphtheria. When it occurs in such diseases as rhachitis and tuberculosis, or when the individual has not been well cared for, the prognosis is also very unfavorable. The prognosis is almost fatal in cases in which the bacillus of Friedländer is the exciting cause, and in other forms when complicated with tuberculosis.

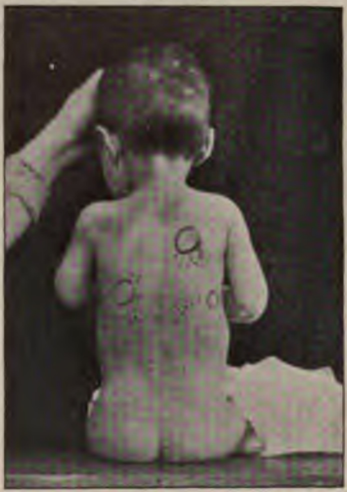
I have already referred to the temperature as a prognostic sign in broncho-pneumonia. According to Holt's observations, the highest mortality occurs among the cases of shortest duration, and the disease is universally fatal when its duration is shorter than four days. After this early period of danger is passed the prognosis becomes much more favorable, the lowest death-rate in Holt's cases being met with in cases terminating in from eight to fourteen days. When the disease lasts for more than two weeks the chances of recovery are lessened every day that the temperature remains raised. The cases in which there is a very high temperature, 41.1° C. (106° F.), are usually fatal. When the disease is protracted, death generally occurs from exhaustion.

TREATMENT.—The treatment of broncho-pneumonia is that of the special disease to which it is secondary, and is for the most part symptomatic. The strength should be carefully supported from the time that the disease is first detected until convalescence has been completely established. The patient should be intelligently nursed, as the nursing is the most important part of the treatment. The atmosphere of the room should be equable, the temperature from 20° to 21.1° C. (68° to 70° F.), and especial attention should be paid to the ventilation. The heat and ventilation obtained from an open wood-fire are especially valuable.

As few drugs as possible should be given, since there is no remedy which will cut short the disease, and most of the drugs commonly used in the treatment of pulmonary affections are, as a rule, of more harm than benefit in broncho-pneumonia. The vitality of infants and young children is so easily lessened by a disease so severe as broncho-pneumonia that the respiratory power is likewise quickly diminished, and we should avoid, except when they are especially needed, such drugs as opium. Ipecac in minute doses seems to facilitate the removal of the mucus. During severe paroxysms an atmosphere of steam or the administration of oxygen is indicated, according as the symptoms seem to be produced by a tenacious exudate or by unaërated lung-tissue. Oxygen is also of benefit in relieving the cyanosis and dyspnœa throughout the disease, and may be given for ten minutes every one or two hours. In cases in which cyanosis and dyspnœa are urgent, if the symptoms depend upon mechanical obstruction with its resulting atelectasis, an emetic is occasionally demanded. In some cases, also, when much exhaustion arises from incessant coughing, small doses of tinctura opii camphorata may be used with caution, and discontinued as soon as possible. When the urgent symptoms are caused by the heightened temperature, much relief can be obtained by means of a bath given at a temperature of 32.2° C. (90° F.). This may be followed by the warm wet pack, which can often be continued with benefit for several hours, and is especially beneficial in producing deep inspirations by which dependent portions of the lung are aërated. In some cases reaction does not take place, and signs of exhaustion follow the bath. Under these circumstances phenacetine may be substituted for the bath. The dose should be 0.06 to 0.12 gramme (1 to 2 grains), guarded with five or ten minims of brandy. This will often reduce the temperature two or three degrees, and in some cases it will remain down for five or ten hours, during which period the infant shows a marked diminution of its nervous symptoms. The position of the child should be changed from time to time, as there is a tendency for the inflammatory exudate to collect in the lower and back portions of the lungs. The administration of food at regular intervals is very important, and should be carried out rigorously.

The chief part of the diet, if possible, should be milk, supplemented with broths, the food being regulated on the general principles governing feeding at different ages. Although vomiting may occur in certain cases, as a rule, if the diet is carefully regulated and the milk given once in two hours with stimulants adapted to the condition of the especial case, an over-sensitive condition of the stomach is seldom a serious obstacle to the treatment.

FIG. 148.



Acute broncho-pneumonia. Female, 4½ years old. The black circles indicate areas of consolidated lung-tissue; the black spots indicate râles.

As the disease progresses it is quite common for the infants to refuse their food. Feeding by means of the stomach-tube, preferably through the mouth, then becomes imperative.

The paroxysmal attacks of cyanosis and dyspnœa may be caused by a weak heart. In these cases the administration of brandy and digitalis, the latter in the form either of tincture or of infusion, for a few days, until the cardiac condition has improved, is indicated. Strychnine and nitroglycerin may also be used, the former being considered especially important. Appropriate doses of alcohol for children of different ages is given on page 456, and those of the other more important cardiac stimulants on page 470.

When convalescence has been established the children are often left in a very weak condition, and special attention should then be paid to the nursing and to the general hygiene. The restoration of strength should be aided by means of tonics, and, if possible, the child should be removed to an equable, warm climate.

Fig. 148 represents a girl, four and one-half years old, in whom the physical examination showed very marked lesions of broncho-pneumonia.

There was no tubercular history in her family. She had scarlet fever when she was one year old, measles when she was one and a half, pertussis and varicella when she was three, and bronchitis when she was three and a half years old. She had otherwise always been well, when she began to complain of headache and pain in her chest. On the ninth day of the attack she vomited, and two days later began to cough and to be rather somnolent. Her bowels were regular. On physical examination the child was found to be rhachitic, as shown by a rosary, enlarged epiphyses of the wrists and ankles, and marked bowing of the legs. On entering the hospital her pulse was 160, her respirations 60, and her temperature 39.4° C. (103° F.) in the morning and

40° C. (104° F.) in the evening. She seemed very sick, had considerable cough, but no expectoration; there was some dyspnœa, and at times she was somewhat cyanotic. On examining the chest the percussion was found to be resonant, but throughout both lungs there were moist râles. Nothing abnormal was detected on examining the heart and abdomen. She was treated with milk and brandy.

On the following day she was in about the same condition, and her pulse, respirations, and temperature were the same as when she entered the hospital. In certain circumscribed areas in both backs slight dulness was detected on percussion, with moist râles around the edges of these areas.

On the third day the pulse had fallen to 136, the respirations to 40, and the temperature to 38.3° C. (101° F.). On the ninth day of the disease the pulse was stronger and the child's condition was very much improved. The dyspnœa had disappeared almost entirely, there was no cyanosis, and she was more comfortable. The front of the chest showed the resonance on percussion to be normal, and no abnormal sounds on auscultation. On examining the back certain circumscribed areas of dulness were found, the borders of which are marked in black. One of these areas was between the edge of the scapula and the vertebral column, another was at the right base in the posterior axillary region, and another at the left base just below the angle of the scapula. Over these areas of dulness bronchial respiration was heard. Just outside of the areas of dulness moist râles of various sizes, which are indicated by black dots, could be heard in limited areas. The physical signs corresponded in their distribution to the areas in which the lesions of this disease are usually detected on physical examination.

On the fourteenth day the child was found to have much improved. During the following ten days the abnormal signs in the chest disappeared, but the pulse, respirations, and temperature did not become permanently normal for a week later. The child, after remaining weak and debilitated for some time, was finally discharged from the hospital in good condition. The temperature, pulse, and respirations declined by lysis on the ninth day of the disease and reached the normal on the twelfth day.

This case apparently arose in the course of a slight bronchitis occurring in a rhachitic child. The prognosis of broncho-pneumonia in rhachitis is usually unfavorable, but in this case the child possessed sufficient vitality not to succumb to the disease.

The following case illustrates the gravity of broncho-pneumonia when it occurs in a rhachitic child.

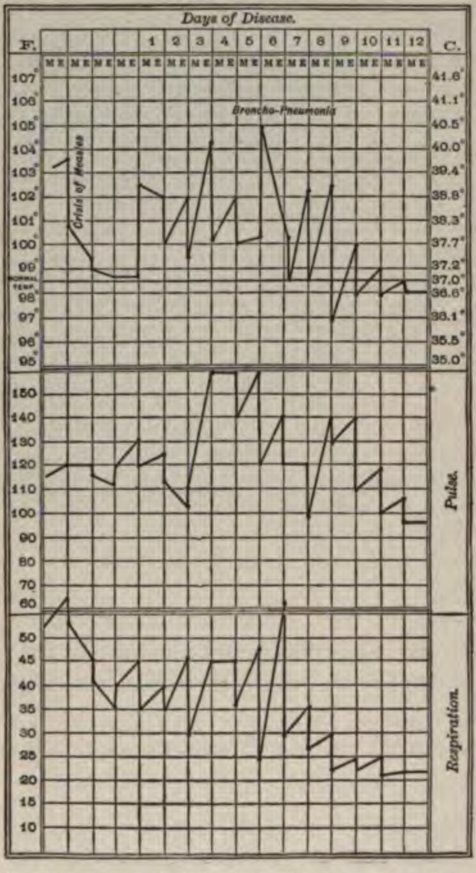
The child was two years and one month old. Its mother died of pulmonary tuberculosis. It had bronchitis when it was one year old, and the cough continued for three months. One week before entering the hospital it was attacked with a severe cough, and began to lose in weight and to have diarrhœa. A physical examination showed that it was a case of marked rhachitis. The breathing was rapid and labored, there was considerable cyanosis, and the child was dull and somnolent. Patches of dulness were found in various parts of the lungs, with moist râles of different sizes. The pulse varied from 140 to 150, the respirations from 80 to 90, and the temperature from 38.8° to 40° C. (102° to 104° F.). The symptoms increased in severity, the child grew weaker, and on the second day after it entered the hospital it died suddenly.

When broncho-pneumonia attacks a child with such marked rhachitis as was shown in this case, a fatal issue almost always results.

The next case was that of a girl, two years old, who previously had been well and strong until she was attacked with measles. There was no tubercular history in the family. On the fifth day of the measles her temperature was 39.8° C. (103.7° F.), the pulse was 120, and the respirations were 62. An examination of the chest showed the heart to be normal, the percussion normal, and the respiration harsh, with numerous fine and coarse moist râles throughout both lungs. Nothing else abnormal was detected.

The temperature fell by a sharp crisis on the sixth day and remained normal for two days, during which the efflorescence rapidly faded and the patient seemed better, though she occasionally had a harsh cough. On the ninth and tenth days she had a slight rise of temperature, but there were no marked symptoms until the eleventh day, when the temperature rose to 40° C. (104° F.), the pulse to 160, and the respirations to 50. She was very restless, and had considerable dyspnœa. She was pallid and sometimes slightly cyanotic. On physical examination there was found diminished resonance over an area in the lower part of the left back. Over this area the breathing was bronchial, and there was also a number of moist râles. On the right side of the thorax, especially at the base of the lung, there were numerous coarse moist râles and harsh respiration, but no dulness. Chart 31 shows the course of the temperature, pulse, and respiration during the twelve days of the broncho-pneumonia. The pulse continued to be rapid and the respirations to be somewhat raised for some days after the temperature became normal. The abnormal signs in the chest disappeared, and the child made a rapid recovery.

CHART 31.



Broncho-pneumonia following crisis of measles. Child, 2 years old.

This case illustrates the rapid development of a broncho-pneumonia during an attack of measles, occurring after the temperature produced by measles had fallen to the normal and while the efflorescence was disappearing. The physical signs showed the presence of small areas of consolidation in the left lung, and the usual diffuse bronchitis throughout the right lung and parts of the left lung.

Chronic Broncho-Pneumonia.—In a certain number of cases, after a child has had an attack of acute broncho-pneumonia the physical signs of consolidation may persist, although apparent recovery has taken place so far as the general symptoms are concerned. When this occurs the fever may return after a variable period, and the child, after having become still more emaciated, may die after a number of months of exhaustion.

These cases are very apt to be associated with a tubercular broncho-pneumonia which may eventually heal by the production of fibroid cicatrices, but the most common termination of these cases is in acute general miliary tuberculosis. Instead of this fatal issue, the child, as has been shown by Delafield, may be left with a chronic form of the disease, which may last for many years and be accompanied by symptoms of cough, dyspnœa, and at times periods of fever. Broncho-pneumonia of a subacute or of a chronic type is so apt to develop in the lungs of young children during the course of any disease of a prolonged nature that frequent examinations of the lungs should be made, in order that the insidious development of these pulmonary lesions may not be overlooked.

TREATMENT.—The treatment of these chronic cases of broncho-pneumonia is essentially symptomatic and climatic. The child should be taken to a warm, dry climate of high altitude, where it can live in the open air and where it will not be subjected to frequent atmospheric changes.

HYPOSTATIC PNEUMONIA.

Hypostatic pneumonia is an extreme degree of passive congestion which occurs in the course of many long-continued diseases, such as typhoid fever. The dependent portions of the lungs become engorged with blood and the alveoli partially filled with accumulated alveolar cells, so that the condition resembles a consolidation. It occurs especially in patients whose vitality has been greatly lowered by a prolonged illness, and who are kept too long in one position without being turned.

The physical signs are those of slight dulness on percussion, generally at the base of the posterior lobes, diminished tactile fremitus, and feeble respirations and voice-sounds. Moist râles, generally bilateral, are heard over the areas of dulness, but fever, pain, and cough are absent.

The condition is recognized by the presence of the primary disease and by the gradual development of the physical signs without the characteristic features of pneumonia or pleurisy. It may, however, give no evidence of its presence during life, and may be discovered only at the autopsy.

TREATMENT.—The treatment is chiefly prophylactic during the course of the primary disease. The nurse should see that the child is not allowed to lie in one position for more than two hours. The condition calls for active stimulation.

ATELECTASIS.

Atelectasis is a collapsed and unaërated condition of the air-vesicles. It may be congenital or acquired.

Congenital Atelectasis.—Congenital atelectasis arises because the infant has not sufficient general vitality and respiratory power at birth to inflate fully all parts of its lungs. There may be an obstruction by mucus. There are in all these cases areas of uninflated pulmonary vesicles of varying extent. These vesicles at the post-mortem examination can easily be artificially distended and then cannot be distinguished from those which have been normally inflated.

Symptoms.—The symptoms of congenital atelectasis are cyanosis, dyspnœa, rapid respiration, rapid, feeble, and often intermittent pulse, a temperature usually lowered, and dulness on percussion with lessened respiration over the area of atelectasis. These are the typical physical signs, but in many cases some or all of these signs are absent and the condition is detected only at the post-mortem examination.

Acquired Atelectasis.—Acquired atelectasis is a symptom of a number of diseases, and occurs especially in severe cases of bronchitis in which the smaller bronchi are involved, also in broncho-pneumonia and in pertussis. Acquired atelectasis undetected during life is frequently found at the post-mortem examination of infants and young children dying of almost any disease.

Prognosis.—The prognosis in these cases varies according to the extent of the pulmonary tissue involved and the vitality of the infant. As a rule, the prognosis is very unfavorable.

Treatment.—The treatment of atelectasis is to stimulate the infant, and to endeavor to raise its temperature by means of a warm pack. In a number of cases I have found the administration of small quantities of oxygen to be of benefit. Artificial inflation of the air-vesicles has not proved to be an especially valuable form of treatment. Many cases of atelectasis may be avoided if the physician appreciates the importance of making an infant, in the first few minutes after birth, thoroughly expand its lungs by vigorous crying.

EMPHYSEMA.

Emphysema is an over-distention of the alveoli and infundibula of the lungs. It is always a secondary process. Various descriptive terms have been used to denote differences in the type of the disease.

Compensatory emphysema consists in a dilatation of the alveoli in certain portions of the lung as a result of obliteration of the function of other parts by consolidation, adhesions, or pressure of deformed bones. This condition is likely to occur in connection with pneumonia, tuberculosis, chronic adhesive pleurisy, or rhachitis.

Obstructive emphysema represents the same pathological lesions, but the cause lies in some obstruction to expiration, as in stenosis of the larynx, asthma, and pertussis.

Interstitial emphysema occurs rarely as a result of a rupture of the walls of the alveoli, by which the air works through the interstitial tissue of the lung into the subcutaneous tissue.

Symptoms.—Hyperresonance on percussion, feeble breathing, prolonged expiration, diminution in tactile fremitus and voice-sounds are the physical signs of emphysema. The normal area of cardiac dulness may be much diminished by the overlapping emphysematous lung; in the same way the upper border of the liver dulness may be considerably lowered. The symptoms are those of the disease in which the emphysema occurs.

TREATMENT.—The treatment should be adapted to the underlying condition.

GANGRENE AND ABSCESS OF THE LUNG.

Gangrene and abscess of the lungs are represented essentially by the same clinical symptoms, and may occur in connection with lobar or broncho-pneumonias. They are caused by the action of suppurative and putrefactive bacteria which invade the affected portions of the lung with the production of small or large abscesses.

Gangrene is diagnosticated chiefly by the presence of putrid, purulent sputum containing elastic fibres. It is to be distinguished from fetid bronchitis, which does not contain the elastic fibres of the lung-tissue.

The rupture of an empyema into a bronchus, with the resulting expectoration of purulent sputum, simulates the condition of abscess, but the diagnosis should be made by the physical signs of empyema. The treatment is that of the primary disease.

DISEASES OF THE PLEURA.

Inflammations of the pleura may be primary or secondary, acute or chronic, and with or without an exudation.

ACUTE PLEURISY.

ETIOLOGY.—Acute pleurisy, either with a simple exudation of fibrin or accompanied by fluid, is not infrequent in children in the winter and early spring.

Primary forms occur, but are very rare, and are attributed to exposure to cold or to injury, as from penetrating wounds and fractured ribs.

Secondary forms are the rule; they occur by direct extension in connection with lobar pneumonia, broncho-pneumonia, tuberculosis of the lungs, pulmonary embolism, pericarditis, peritonitis, or some neighboring local inflammation. They may also be caused by metastatic infections from distant inflammatory processes, by means of the blood-vessels or lymphatics, or in the course of acute infectious fevers. Pleurisy may also occasionally be associated with nephritis and syphilis.

The bacteriology of inflammations of the pleura includes many varieties of organisms. The pneumococcus is found in the great majority of cases of pleurisy in early life and is generally accompanied by a purulent exudation; these cases are apt to arise in connection with pneumonia and run a favorable course. The streptococcus pyogenes causes a much more serious, and often fatal, purulent infection, but is less common in children than in adults.

Tubercular pleurisy is rare in infancy, but occurs more frequently in the later years of childhood. Eichhorst has shown in one series of serous exudations that nearly sixty-five per cent. of the cases were tubercular, but the pneumococcus, diplococcus, and staphylococcus are sometimes found in these non-purulent exudations. The staphylococcus, gonococcus, Friedländer's bacillus, colon bacillus, and typhoid bacillus have occasionally been cultivated from the purulent exudations.

In acute inflammations of the pleuræ we can make a clinical distinction between three types of the disease, based upon differences in the pathological lesions: (*a*) acute dry or plastic pleurisy, (*b*) acute pleurisy with sero-fibrinous exudation, and (*c*) acute pleurisy with purulent exudation (empyema).

Acute Dry or Plastic Pleurisy.—PATHOLOGY.—The pleura is injected and without lustre. At first it is covered with a thin layer of lymph, which becomes opaque and thickened by the deposition of fibrin and leucocytes. The sub-pleural fibrous tissue is also thickened by the exudation of serum and by cellular infiltration. The roughened surfaces of the visceral and parietal layers may adhere, forming loose connections which may subsequently undergo organization without producing an exudation and end in a chronic adhesive pleurisy. The areas of inflamed pleura may be large or small and diffusely located. They may rarely occur at the apices of the lungs in connection with an early tubercular process. On the other hand, they may be limited to the base and give rise to diaphragmatic pleurisy.

The term *pleuro-pneumonia* has been applied to those cases of dry pleurisy in which there has been an excessive exudation and deposition of pus-cells in the layer of fibrin without the formation of free fluid in the pleural cavity.

SYMPTOMS.—Although localized areas characterized by the production of fibrin are quite frequently found at the post-mortem examination, the diagnosis of this form of disease in infants and in young children is not often made during life. The symptoms are of acute onset, characterized by a sharp pain or stitch in the side, slight fever, and a short, dry cough. The pain is usually referred to the lower part of the axilla, where the largest excursions of the two layers of the pleura take place in the act of breathing, or to the abdomen. On auscultation a friction-rub may be heard at the site of the pain or remote from it. In infants and young children, however, friction-sounds and pleuritic pain are much less pronounced than in adults, and a pleurisy is often not even suspected until the advent of the exudation. Probably in the great majority of cases of dry pleurisy in early life the condition is secondary to lobar or broncho-pneumonia, and its signs and symptoms may be masked by those of the primary disease.

Acute Pleurisy with Sero-Fibrinous Exudation.—PATHOLOGY.—The pathological exudate of a dry or plastic pleurisy may be directly absorbed or the inflammation may result in an exudation of serum and fibrin into the pleural cavities.

The fluid is of a clear, pale-yellow color, coagulates spontaneously, and contains flocculi of fibrin and a few leucocytes and endothelial cells. The amount of fibrin varies, and, according as it is small or large in relation to the serum, we speak of the exudate as sero-fibrinous or fibrino-serous. A greater proportion of fibrin is likely to be present in the exudation in children than in adults.

The quantity of fluid may be small and *encysted*, and accompanied by areas of atelectasis in the adjacent lung, or it may fill the whole cavity, compressing the lung against the spine, and thus practically depriving it of its functions. In a similar manner when the exudation is on the right side the liver is depressed. Displacements of the heart and pericardium very commonly occur when the exudation is either on the right or the left side.

SYMPTOMS.—The onset of acute pleurisy with serous exudation is in many cases violent, and attended by a high temperature, increased respirations, rapid pulse, restlessness, and even pain, which in young children is usually referred to the abdomen. In infants and in young children convulsions are quite common, while in older children the symptoms are more like those which occur in adults. There is a short, painful cough, with loss of appetite, and frequently vomiting and diarrhœa.

The white blood count is rarely increased in serous exudations, except in the febrile stages, and even then is not often over thirteen thousand. Uncomplicated cases of tubercular pleurisy probably never have a leucocytosis (Cabot).

These early symptoms are usually followed in two or three days by an exudation and by a decided lessening of the pain and dyspnœa. At the same time the temperature begins to have a decided morning remission. When the exudation is large, the children lie more comfortably on the affected side, and when they are nursing they nurse most easily from the right breast if the left pleura is affected, and from the left breast if the right pleura is affected. After the serous exudation has remained for a few days it ordinarily begins to lessen in amount, absorption takes place, and by the end of a week or ten days it is entirely absorbed and the child recovers. In other cases it becomes chronic unless its absorption is furthered by aspiration.

PHYSICAL SIGNS.—The physical signs of fluid in the pleural cavity differ in certain respects from the usual signs in adults.

Inspection.—On inspection we find diminished movement of the affected side on inspiration, and if the fluid is large in amount, a decided bulging. The impulse of the apex beat is often displaced, depending on the quantity of the exudation and the side affected. The displacement of the heart is most marked in left-sided effusions, in which the impulse may be seen in the third and fourth right intercostal spaces, and sometimes as far out as the mammillary line. In right-sided effusions the degree of displacement is less, but the impulse may be seen in the fourth interspace and between the mammillary and midaxillary lines.

Palpation.—By palpation the intensity of the apex beat and the amount of displacement of the heart and of the liver (in right-sided affections) may be determined. The information to be obtained by tactile fremitus is not of much value in infancy and early childhood; its diminution is not marked, and occasionally it is quite distinct over an exudation. Thus one of the most valuable of differential signs from consolidation fails in the doubtful cases in children.

Percussion.—The percussion-note varies from slight dulness to flatness, according to the quantity of the fluid, and there is a sense of increased resistance to the fingers. Above this level of dulness the note is normal or tympanitic (Skoda's resonance), and may be most pronounced in front below the clavicles. On the right side the dulness may be continuous with that of the liver, on the left it may obliterate the semilunar space of Traube.

The upper border of a fluid exudate has certain peculiar characteristics, as shown by Ellis and Garland, if not complicated by pleural adhesions, and is often of aid in the differential diagnosis from consolidation of the lung. When the fluid is small in amount it can usually first be detected in the back. In these small exudations the upper border of dulness begins at the vertebral column, extends outward horizontally for a distance which varies according to the size of the exudation, and drops in the neighborhood of the posterior axillary line, by a curve more or less abrupt, to the base of the thorax. As the fluid increases in size the line of dulness drops more anteriorly. When it is moderate, the lower half of the pleura only being filled, the upper border of the area of dulness is found to extend at first outward and then upward over the angle of the scapula, reaching its highest point in the axillary region. The line may then drop abruptly from the upper axilla to the base of the thorax near the apex of the heart or to the corresponding point on the right side. This line, which has been called the "letter S curve," is characterized by having its highest point in the axillary line. When the quantity of fluid is still larger and exceeds a certain amount, the "letter S curve" is obliterated, and the resonance over the compressed lung becomes less marked. This curve can only be determined by very light percussion.

If the line of dulness is altered by a change in position of the body, it is a positive sign of fluid. An encysted exudation or a large amount of fluid will not give this sign.

The displacement of the heart is a most valuable indication of effusion in young children, and with careful, light percussion the gradual increase and decrease of the effusion, when it is of any great extent, can be determined by the variations in the position of the heart. The physiological dulness of the heart which has been described as occurring in early childhood (page 90) under the lower third of the sternum, becomes much more marked when the heart is displaced.

Auscultation.—The sounds obtained by auscultation are variable, and in some respects are very different from those which are heard over a pleural exudation in an adult. Bronchial breathing is the rule rather than the exception. It is, however, less intense and more distant than that which we ordinarily hear over an area of consolidation. Vocal resonance, moreover, is generally increased (bronchophony), and is sometimes as pronounced as in pneumonia. Occasionally it will give the same nasal quality (egophony) as over a consolidation. Friction-sounds above the level of the fluid, or after absorption, are rare in early life. Moist râles are sometimes heard over an exudation, and are probably of bronchial origin. Even friction-sounds are sometimes said to be heard over the fluid, but are probably transmitted from points above the level of the exudation.

Diagnosis.—The principal physical signs of pleural effusion in infancy and early childhood are flatness which does not correspond to the anatomical divisions of the lobes of the lung, bronchial or distant tubular breathing, bronchophony, displacement of the heart, and changes in the outline of dulness on shifting the position of the body. The differential diagnosis can best be described in connection with purulent exudations (page 713).

Prognosis.—The prognosis of a serous effusion, as a rule, is very favorable in infants and in young children unless the streptococcus or one of the more virulent forms of the pyogenic cocci is present, or unless the disease is secondary to tuberculosis elsewhere and is caused by the tubercle bacillus. If the serous exudation tends to become purulent, the prognosis is not so good, but still, provided appropriate treatment is carried out, it is favorable. If, as in rare cases, the pleuritic exudation occurs on both sides, the prognosis becomes grave. The possibility of the presence of the tubercle bacillus should be considered in these latter cases.

I had in my service at the Boston City Hospital a boy, thirteen years old, who was attacked with pleurisy and a serous exudation on the left side with displacement of the heart to the right. After one aspiration the fluid was quickly absorbed, but three weeks later he was attacked with pleurisy on the right side, followed by an exudation and displacement of the heart to the left. This effusion was absorbed without aspiration, and the boy was discharged from the hospital well and strong, with both lungs apparently in a normal condition.

When the effusion is very large and the heart is much displaced, there is always the danger of a fatal issue from asphyxia, and the prognosis depends upon whether the exudation can be controlled by aspiration and the heart thus be kept in normal position. The following case illustrates the danger which may arise when these large quantities of fluid are accompanied by displacement of the heart.

A boy, four or five years old, entered the hospital with a large exudation in the left chest. The heart was displaced to the right and upward as far as the second interspace to the right of the sternum. He was cyanotic and gasping. On aspirating the chest and removing a large quantity of fluid, the heart reassumed its normal position under the sternum. On the following night the boy died suddenly, the fluid having rapidly reaccumulated and again displaced the heart.

Cases of this kind should warn us that a pleuritic exudation of any extent in a young child should be watched carefully, and that aspiration should be performed when there is indication of an increase in the intrathoracic pressure.

When the pleurisy is secondary to other diseases, such as rheumatism and scarlet fever, the prognosis is not so favorable; the fluid is not apt to be absorbed so readily, and is more likely to become purulent. The prognosis is also rendered more unfavorable in these cases by the prolonged pressure upon the lung, with its corresponding ill effects upon the general condition of the child. The dangers which arise from the development of tuberculosis must also be borne in mind.

TREATMENT.—The treatment of acute pleurisy during the early days of the attack, before an exudation of any considerable extent has appeared, should be directed to the relief of the pain by a flannel bandage closely applied to the thorax, so as to allow the ribs to move as little as possible in respiration. Sometimes an occasional dose of tinctura opii camphorata will also be needed to make the child comfortable. After the fluid has increased, the child should, if possible, be kept in bed. There are some cases, however, in which a child with considerable exudation in its pleura will feel well and bright, and will play about its nursery without showing any especial symptoms of discomfort. I have met with instances of this kind where, except for pallor and a poor appetite, the child seemed bright and active, and yet had a pleuritic exudation large enough to displace the heart.

The child should be examined each day in reference to an increase in the quantity of fluid and to a displacement of the heart. If neither occurs, and if the respiration and circulation show no evidence of disturbance, an expectant treatment is all that is required. If, on the contrary, the fluid is increasing and the heart is displaced to any considerable degree, or if absorption of the fluid is delayed for two or three weeks, the chest should be aspirated. The point of aspiration should usually be in the fourth or fifth interspace in the midaxillary or posterior axillary line, but in deciding where to enter we should always be guided by the indications given by the physical signs. A bacteriological examination of the fluid removed should then be made, to determine which form of organism is present in the exudate. If one of the more benign forms of bacteria is present, such as the pneumococcus, or if the fluid is found to be serous, no further treatment will be required, unless there be a reaccumulation of the fluid, in which case a second aspiration will be indicated. If, however, streptococci are found in the exudate, the case must be watched very carefully, as it is more likely to become purulent and to need radical surgical treatment.

As the unfavorable symptoms in a pleuritic effusion arise mostly from intra-thoracic pressure, relief from the pressure by aspiration is indicated rather than by the use of drugs, which cannot be depended upon.

Acute Pleurisy with Purulent Exudation.—Empyema.—In the first three or four years of life purulent exudations into the pleura are much more common than serous or sero-fibrinous exudations. Nearly all the cases which are secondary to lobar or broncho-pneumonia, representing the largest group in children, are caused by the pneumococcus. The presence of other organisms has already been referred to.

PATHOLOGY.—The inflammatory process begins with an exudation of fibrin upon the surface of the pleura, accompanied by an excessive migration of leucocytes and a rapid accumulation of pus. The exudation may occasionally be sero-fibrinous at first and subsequently become purulent. In cases of streptococcus and staphylococcus infections, the inflammation may be purulent from the beginning, but the pus is not so thick and contains less fibrin than in pneumococcus infections. The pus may be encysted, but this rarely occurs in an empyema following pneumonia.

When no bacteria are found in the fluid, when there is no history of a preceding acute pneumonia or a neoplasm of any kind, when there is little tendency to absorption of the exudate, and when the exudate is found to contain blood, the signs point strongly towards a tubercular origin.

SYMPTOMS.—The disease when primary may be acute in its onset, and may simulate closely the initial stage of lobar pneumonia. When secondary to pneumonia or some other acute infectious disease it is slow and somewhat insidious in its development. The pulse and respirations may be increased, but after the early days of the disease they are often very little raised. There is nothing characteristic in the temperature of an empyema, and the diagnosis usually can be made only from the knowledge that the younger the individual the more likely is pus to be present. Cachexia, anæmia, and prostration soon become prominent. A pronounced leucocytosis is present from the onset and is the most important symptom in the differential diagnosis from a purely sero-fibrinous exudation, which rarely shows more than a moderate increase in the white cells. Leucocytosis may at times be absent when aspiration shows pus to be present, but this is unusual and may be only temporary.

The physical signs are the same as in sero-fibrinous exudations and have been described on page 709.

The absorption of a purulent exudate without surgical interference is very rare. I have occasionally met with cases in which one or two aspirations were all that were necessary and in which complete absorption seemingly took place.

When cases of empyema are left untreated, a spontaneous opening usually takes place through some portion of the thoracic walls, but the exudate may also find its exit through the lungs by opening into one of the bronchi or perforating in other directions. I have met with cases in which the diaphragm was perforated and the point of exit of the pus was in the region of the umbilicus. When perforation does not occur, the pus is partially absorbed, adhesions are formed, and sometimes great deformity of the chest follows, which may result in a marked degree of lateral curvature of the spine as well as in great contraction of the chest.

DIAGNOSIS.—The diagnosis of empyema rests upon the signs of fluid in the pleural cavity associated with a pronounced leucocytosis, but it cannot be made definitely without the aid of the aspirator. In all doubtful cases the aspirator should be freely used, for the consequences of a neglected empyema are very serious.

The differential diagnosis of empyema is to be made from a number of different lesions.

From Pneumonia.—The conditions which are most likely to be confounded with a pleural exudation are acute and unresolved pneumonias. The difficulty arises from the frequency of bronchial breathing and bronchophony and normal tactile fremitus over a pleural exudation in early life. When the physical signs are those both of fluid and of consolidation, the diagnosis must rest upon the results of exploratory aspiration. It is to be borne in mind, however, that aspiration frequently yields a negative result even when fluid is present, and several punctures may be necessary before the diagnosis can be settled. The possibility of pneumonia and pleurisy with exudation being combined should not be forgotten in making the differential diagnosis.

From Atelectasis.—A simple bronchitis in children may cause an occlusion of the bronchi and produce atelectasis of the lungs, the signs of which may simulate closely a small effusion. Coughing and deep inspiration will, however, often remove the mucous plugs and allow the air to enter, so that the condition is readily determined. If the atelectasis persists, the signs may suggest an encapsulated empyema, and aspiration must be resorted to.

From Sero-Fibrinous Exudations.—The diagnosis from sero-fibrinous exudations is very difficult, but the younger the individual the more likely is the fluid to be purulent. After the first week or ten days of the disease, however, when the fluid is purulent, the usual signs of absorption which so commonly occur in a serous exudation are not ordinarily found, and aspiration of the pleural cavity will then determine which form of the disease is present. Marked leucocytosis favors the diagnosis of empyema.

From Chronic Adhesive Pleurisy.—The signs of chronic adhesive pleurisy may exactly resemble those of fluid. If the diagnosis is doubtful aspiration will make it clear.

PROGNOSIS.—The prognosis in empyema is good if the diagnosis is made early, if the pneumococcus is the cause, and if the case is properly treated. If the infection is from the streptococcus pyogenes or the tubercle bacillus the prognosis is very unfavorable. If the pus has been allowed to remain in the chest for a long period, the chances of recovery are proportionately diminished. Spontaneous recovery rarely occurs. In infants under one year the mortality is usually much higher than in later periods, especially in hospital practice.

Treatment.—The treatment of empyema is essentially surgical, and I shall not enter into its details. After the first aspiration, if absorption does not occur within a week and if the infection is due to the pneumococcus, it is well to wait a week before deciding on a second aspiration or operation. If one of the more virulent forms of bacteria is found in the exudate, a radical operation is the best method of treatment and should be performed at once. Sometimes one aspiration of the pus will be followed by permanent and entire recovery. This, probably, only occurs in infections due to the pneumococcus, and I have seen such a case in an infant seven weeks old. I have also seen recovery follow after two and even three aspirations. The pleural cavity should be thoroughly drained by means of drainage-tubes. In many cases, especially in children over two or three years of age, resection of one or two ribs gives the best results. Although in some cases a rapid cure in two or three weeks follows the operation, yet the recovery is often prolonged for many months, even when strict antiseptic precautions have been taken at the time of the operation.

The following cases illustrate pleurisy with sero-fibrinous exudation:

A girl, eleven years old, was attacked with a chill followed by vomiting and a short, dry cough. Later she complained of pain in the lower part of the right chest. She was feverish, lost in weight and in appetite, and her respirations were painful. She lay most comfortably on her back and on her left side. A pleuritic friction-rub was heard in the right axillary region. Her lips and cheeks were slightly cyanotic. Her tongue was somewhat coated. The alæ nasi were working and orthopnœa was marked. The percussion and auscultation of the left lung showed nothing abnormal. The resonance was fair over the upper part of the right front and back. There was flatness from about the fifth dorsal vertebra in the right back to the base of the lung. The flatness extended into the axillary region, where it reached its highest point, and then gradually descended to the right parasternal line on a level with the fourth costal cartilage. Over this area of flatness respiration was markedly diminished. No friction-rub was heard. The vocal and the tactile fremitus were diminished. The impulse of the heart was found in the fourth interspace, 1 cm. (⅜ inch) to the left of the mammillary line. The heart-sounds were normal. There was no displacement of the liver. An examination of the urine showed it to be acid, to have a specific gravity of 1022, to be of normal color, and to contain no albumin. The chlorides were normal. The temperature was 38.3° to 40° C. (103° to 104° F.) for the first two days and then gradually declined to normal in the course of three weeks. The pulse and respirations were irregular, the former ranging between 80 and 120, and the latter between 25 and 50. The physical signs were those of a pleuritic effusion of the right side with displacement of the heart to the left.

The area of flatness gradually decreased, and an exploratory aspiration showed the fluid to be serous. Nine weeks from the beginning of the attack, the dulness on percussion gradually disappeared, auscultation showed the respiration to be normal, and the heart resumed its normal position.

In another case of serous effusion in the pleura, the temperature ranged between 37.2° and 38.8° C. (99° and 102° F.) for three weeks and suddenly dropped to normal. In the beginning 165 c.c. (5½ ounces) of fluid were withdrawn from the chest. The fluid reaccumulated, so that absolute dulness was found over the whole right side of the chest in front and behind, but aspiration did not have to be resorted to again, and complete absorption took place thirty days from the beginning of the attack.

Another case which illustrates the difficulty in diagnosticating a purulent effusion in the pleura in the early days of the disease is the following:

A girl, four years old, was suddenly attacked with cough, and pain in the right side. The temperature was 40.5° C. (105° F.). The respirations were quickened, and the pulse was rapid. Nothing abnormal was detected on physical examination. On the following day the general symptoms disappeared, and the temperature fell to 38.8° C. (102° F.). In another day the temperature fell to 37° C. (98.6° F.), and the child seemed bright and well. On the following day, however, the temperature rose to 40° C. (104° F.), flatness and the other signs of fluid were detected in the right axillary region, and an exploratory aspiration showed the presence of pus.

CHRONIC PLEURISY.

Chronic pleurisy results from previous attacks of acute inflammations of the pleura, either with or without the formation of fluid. The result is essentially the same, differing chiefly in degree. The lesions are represented by adhesions between the thickened layers of parietal and visceral pleura. Large or small areas of the cavity may thus be obliterated. The physical signs are represented chiefly by dulness and diminution in the intensity of the breathing, voice-sounds, and tactile fremitus. Litten's phenomenon is absent. On the other hand, adhesions are often found at the autopsy, where during life both lungs seemed perfectly normal. The diagnosis is aided by the use of an aspirating needle, by which we can often determine by the sense of touch whether the needle is in a thickened pleura, the lung, or a free cavity. If the adhesions are extensive, retraction of the chest with certain compensatory changes occur as illustrated in the following case.

A little girl, three years old, had an attack two years previously of some pulmonary disease accompanied by fever. From that time until she was first seen she was delicate and coughed a great deal. Her cough had increased, but she did not lose in weight nor have any other abnormal symptoms. She was pale, and the cervical, axillary, and inguinal glands were enlarged. Her fingers were markedly clubbed. She showed a peculiar lateral curvature of the spine, which could not be made to disappear by traction. The right side of the thorax expanded normally, the left side scarcely at all. There were hyperresonance, no râles, and compensatory respiration over the right lung. The left lung was apparently atelectatic and showed dulness everywhere except in a small triangular area at the inferior angle of the scapula. This deformity of the thorax was probably the result of an empyema which occurred when she was one year old and was not properly treated.

Treatment.—The treatment consists entirely of general hygiene and systematic exercises of the chest.

HYDROTHORAX.

Hydrothorax is a non-inflammatory affection, characterized by the transudation of a simple fluid into the pleural cavities. It is a rare affection in children. The effusion is the result of mechanical obstruction to the flow of the subpleural blood and lymph. The principal causes for this obstruction are found in diseases of the heart and kidneys. The fluid is generally bilateral; in some cardiac cases it may be unilateral. It is apt to be greater in amount on one side than on the other.

The transudation is watery in character, of low specific gravity, under 1015, and contains less albumin than inflammatory exudations, generally from one to two per cent. It does not coagulate spontaneously unless complicated with the exudation of an inflamed pleura. A few leucocytes, red blood-corpuscles, and endothelial cells may be found when the sediment obtained by the centrifuge is examined microscopically.

Symptoms.—The symptoms are in most cases simply those of the condition to which the hydrothorax is secondary. If the fluid is excessive there is a sense of constriction beneath the sternum, dyspnœa, and short, rapid respirations, weak pulse, and sometimes cyanosis. Any of these symptoms may be due to the primary disease alone, and it is therefore of much importance to watch the lungs carefully in all advanced and serious cases of cardiac and renal disease associated with dropsy.

The physical signs are those of pleurisy with exudation. There is no friction-rub, as the pleuræ are not inflamed, and the fluid shifts more readily on change in position, because of the absence of inflammatory adhesions.

Diagnosis.—The diagnosis can be made from inflammatory exudations by the chemical examination of the fluid obtained by aspiration.

Treatment.—The treatment is that of the primary disease to which the hydrothorax is secondary. If the symptoms of pressure are serious, repeated aspiration is generally necessary.

PNEUMOTHORAX.

Pneumothorax, or air in the pleural cavity, rarely occurs alone. It is generally associated with serous or sero-fibrinous fluid, and is then called *hydropneumothorax;* or with pus, when we speak of it as *pyopneumothorax.*

Etiology.—Air may enter the pleural cavity from penetrating wounds from without, such as may result from aspiration. The most frequent cause, however, is due to perforation of the visceral pleura in the course of pulmonary tuberculosis, gangrene, or abscess. Nine-tenths of the cases, according to Fraentzel, are due to pulmonary tuberculosis. The condition is of rare occurrence in children.

Pathology.—As a result of the entrance of air into the pleural cavity, the lung collapses and is withdrawn into the upper and posterior part of the pleural cavity. The amount of retraction depends upon the extent of the pleural adhesions or consolidation present. The entrance of air is generally accompanied by micro-organisms, and by the conditions which follow a sero-fibrinous or purulent inflammation of the pleura.

SYMPTOMS.—The onset when sudden is characterized by severe pain in the side, dyspnœa, a weak, rapid pulse, and great prostration. Sometimes the pneumothorax develops insidiously without giving rise to these symptoms of distress. The physical signs are distinctive.

Inspection.—Respiration is diminished on the affected side and Litten's sign is absent. The heart may be displaced in the same way as by a pleuritic effusion.

Palpation.—Tactile fremitus is absent over the lower part of the chest, but may be normal or even increased at the upper posterior portion over the retracted lung. The liver is generally displaced if the pneumothorax is on the right side. The apex of the heart will also be found to be displaced as in effusions.

Percussion.—Tympanitic resonance is pronounced throughout the affected side, and may be so marked as to mask the presence of small effusions. When the air within the pleural cavity is very tense, the percussion-note may be muffled, simulating dulness.

Auscultation.—The respirations and voice-sounds are very faintly heard or are absent in the lower portions of the chest, whereas at the top a faint amphoric or metallic breathing may be heard. If one coin is tapped against another coin placed on the back over the tympanitic area, the metallic echo is readily transmitted to the stethoscope on the front of the chest, and is quite characteristic. When the pneumothorax is accompanied by fluid, as is generally the case, the signs of pneumothorax and fluid are combined and vary in their character according to the relative predominance of fluid or air. The sound of splashing obtained by succussion is very distinctive of air and fluid, but must not be confused with similar sounds transmitted from the stomach. The difference can be made out by attention to the position of the intensity of the sound.

DIAGNOSIS.—Only when the air is very tense, and gives a muffled or dull note, will the physical signs resemble those of fluid. If the diagnosis is doubtful, aspiration will make it clear. Very large tubercular cavities may give rise to the signs of pneumothorax, but they are rare in children, and even when present are not associated with succussion or displacement of organs.

Pneumothorax and emphysema resemble each other in the signs of feeble breathing and hyperresonance, but emphysema is almost always bilateral, with the coarse moist râles of an associated bronchitis, and does not displace the heart or liver, although it may alter the cardiac and hepatic areas of dulness.

PROGNOSIS.—The prognosis of pneumothorax depends largely upon the primary condition to which it is secondary.

TREATMENT.—The treatment is essentially the same as that of pleurisy with effusion.

DIVISION XI.

DISEASES OF THE HEART AND PERICARDIUM.

DISEASES OF THE HEART.

CARDIAC disease in infancy and early childhood may be divided into congenital or acquired, developmental or inflammatory, organic or functional, acute or chronic. In this early period of life cardiac disease has certain characteristics in which it differs essentially from those which are met with in later life. One of these characteristics is that there is a more decided tendency to recovery than at a later period. Another is that, owing to the undeveloped condition of the infant and young child, interference with the growth of other organs and parts of the body may more easily result from diseases connected with the circulation than is possible in the case of the fully developed adult. Thus, there are certain anatomical facts connected with the ossification of the sternum which become of great importance in connection with cardiac disease. Deformities of the thorax may result from the continued pressure of the enlarged heart on the soft and pliant sternum and costal cartilages of the young subject. These deformities do not arise merely when the individual is rhachitic, but may also depend upon the stage of development at which the cardiac disease begins. The deformity is more or less pronounced in inverse ratio to the age and in direct ratio to the time during which the cardiac disease has existed. The shape and extent of the deformity are also dependent upon the degree of ossification which has taken place in the sternum.

In young infants, in whom the entire sternum is in a cartilaginous condition, the intra-thoracic pressure from an enlarged heart may cause a bulging of the whole front of the thorax. This may occur during the first year, and even up to the third year. As the child grows older, the manubrium and the second piece of the sternum become ossified and offer more resistance, while the third piece of the sternum, still remaining in a semi-cartilaginous condition, may be tilted. This latter displacement may occur in children in whom the cardiac disease has not developed until the fourth, fifth, or sixth year. I have had under my care a child seven years old who at the age of five years had articular rheumatism with resulting cardiac hypertrophy, and who presented this displacement of the third piece of the sternum. No other signs of rhachitis were detected. The middle period of childhood is also a peculiarly unfortunate one for the occurrence of cardiac disease, because the heart grows so rapidly at this period that it requires a proportionately greater amount of intra-thoracic space for the normal performance of its function than it does later.

In addition to the injury which may be done to the thoracic walls by an enlarged heart, we must consider the interference with the normal uniform expansion so necessary for the growing pulmonary tissue and the consequent loss of the elasticity which plays so prominent a part in the establishment of the equipoise which should exist in a perfected respiratory apparatus.

The occurrence of *diseases of the blood-vessels* is rare in infancy and early childhood in comparison with later life. *Aneurism* is rare. A *narrowing of the isthmus aortæ* is more common, and is one of the most marked of the congenital defects of the blood-vessels. It may result in increased blood-tension, followed by hypertrophy of the left ventricle and subsequent dilatation. Sometimes there is an *absence of the isthmus aortæ* during fœtal life. The compensation for this defect takes place by an increased action of the left ventricle and the establishment of a collateral circulation between the subclavian artery and the thoracic and the abdominal aorta. These malformations exert in varying degrees an influence on the heart, as the infant grows older, from increased blood-pressure.

CONGENITAL DISEASES OF THE HEART.

Congenital diseases of the heart are somewhat obscure in their etiology, but usually result either from an interference with the normal development of the organ or from endocarditis, or from a combination of both. The parts of the fœtal circulation at birth which are of most importance in reference to diseased conditions of the heart and great blood-vessels are the foramen ovale and the ductus arteriosus. When these remains of the fœtal circulation, which are normal during intra-uterine life and for a short period afterwards, continue as the infant grows older, they become abnormal and interfere with the equilibrium of the circulation.

When the development of the heart has been interfered with in intra-uterine life, there results another set of malformations, the chief of which are an open ventricular septum, a transposition of the great vessels connected with the heart, and various malformations of the valves, such as atresia of the orifice, absence of one or more cusps, or supernumerary segments. There may also be absence of one of the large vessels. When, again, an inflammatory condition has taken place in intra-uterine life (fœtal endocarditis), various other morbid conditions result, usually located in the right side of the heart, the most common of which are connected with the pulmonary artery, causing stenosis or atresia, a narrowing of the conus arteriosus, and various malformations of the tricuspid valve and other orifices of the heart. These inflammatory lesions of the valves result in dilatation and hypertrophy of the heart and prevent its normal development by keeping open, for the purpose of compensation, the foramen ovale, the ventricular septum, and the ductus arteriosus. The valvular lesions vary greatly in their extent and pathology.

It is sometimes very difficult to distinguish between the lesions resulting from fœtal endocarditis and errors of development. The nodules of Albini and the small hemorrhages which are so commonly found on the cardiac valves in children, must not be mistaken for endocarditis. The form of inflammation of the endocardium which occurs in intra-uterine life is the chronic or sclerotic variety. Verrucose endocarditis is rare.

A deficient filling of the left side of the heart in early life, such as occurs in cases of atelectasis, fœtal pneumonia, or fœtal endocarditis, especially when stenosis of the pulmonary artery has resulted, may delay the closure of the foramen ovale and of the ductus arteriosus, which under these circumstances act as safety-valves. This is true also of the delay in the closing of the intra-ventricular septum, which is often of great aid in preserving the equilibrium of the circulation. The most common congenital cardiac lesions are an affection of the pulmonary artery, an open foramen ovale, an open ventricular septum, and an open ductus ateriosus.

Transpositions of the aorta and pulmonary artery are very commonly met with in connection with other congenital defects, such as spina bifida or hydrocephalus, but may occur in infants who are otherwise normally developed. In these cases the duration of life is almost invariably short.

Although these various abnormal conditions may be found alone, yet they generally occur in combination with each other, and all kinds of transpositions and malformations of the vessels are at times met with.

There are various other malformations of the heart which occur at an early period of fœtal development, and which are of pathological rather than clinical interest. Of these can be mentioned cases in which there are one auricle and one ventricle (*cor biloculare*), or one ventricle and two auricles (*cor triloculare*), as well as a case which has come under my notice, in which the heart had a double apex, the right apex lying in the fourth interspace to the right of the sternum, and the left apex lying in the fourth interspace to the left of the sternum. At times there may be a double heart, absence of heart (*acardia*), heart on the right side (*dextrocardia*), or the heart may be in various parts of the thorax or abdomen.

General Symptoms.—Although in some cases the symptoms of congenital cardiac disease are very indefinite, and the disease may be masked for a number of months, yet in a large number of cases they soon become evident. The typical symptoms of congenital cardiac disease are cyanosis and attacks of dyspnœa, amounting at times to suffocation, and atrophy. As the disease progresses, the fingers and toes often become club-shaped, the nails blue, and the skin cool. In connection with these rational signs there is usually an evident pulsation in the cardiac region, with bulging of the precordia. When the obstruction caused by the lesions is sufficient to produce hypertrophy and dilatation of the heart, an increase in the area of cardiac dulness is found. Diffuse cardiac murmurs are heard often over the whole chest, but usually have their maximum intensity towards the upper part of the sternum, are commonly systolic in time, and in some cases are accompanied by a thrill.

The most common symptom is cyanosis. Cyanosis may arise from incomplete oxygenation of the blood, and not merely from a mixture of the venous and arterial currents. When cyanosis is present to any extent there is usually some malformation of the pulmonary artery or its valves. Well-marked congenital malformations may be present with no symptoms whatever. There may be an entire absence of cyanosis; there may be no increased area of dulness and no murmurs; and I have met with instances in which the infants seemed to be thriving, and showed neither labored breathing nor physical signs of disease up to within a few hours of death, and yet a number of cardiac malformations were found at the autopsy. Although, as a rule, the symptoms occur at a very early period of extra-uterine life, yet quite frequently they are so mild in character that they are not especially noticed, as they may appear only when the infant is much excited or crying. The cardiac symptoms may not be prominent enough to attract attention until the infant is old enough to exert itself sufficiently, as by creeping or walking, to interfere with the equilibrium of its circulation. At times another disease, especially bronchitis or pneumonia, may precipitate the cardiac symptoms. It is quite common for endocarditis to develop in a heart in which a congenital malformation is present. The diagnosis between a congenital and an acquired cardiac affection then becomes necessary and is accompanied by many difficulties.

The following case illustrates how congenital cardiac disease can be masked for a number of weeks:

The infant was apparently healthy at birth, and a careful physical examination showed nothing abnormal in the thorax. There was no cynosis noticed, the skin being of a normal color. When it was sixteen days old it refused to take the breast, and in the afternoon seemed somewhat cold, was slightly cyanotic, and had a temperature of 35.2° C. (95.5° F.). An examination of the heart detected nothing abnormal. A few drops of brandy were given to it, and after several hours the skin became warm, the respirations normal, and it took its food as usual. Early in the following morning the quickened respiration returned, the temperature rose to 37.7° C. (100° F.), it refused to take its food, failed rapidly, and died in the afternoon.

The examination of the heart showed a large open foramen ovale and an absence of the upper part of the intra-ventricular septum below the aortic valve. The beginning of the aorta for a distance of 1 cm. (⅜ inch) was dilated into a spherical pouch, from which were given off (1) the aorta without any branches before the intercostals, thus supplying only the lower part of the body, (2) a large vessel to the right lung, and (3) a large vessel to the left lung. From the upper part of the right ventricle was given off a large vessel which divided 1.4 cm. (½ inch) above the pulmonary valve into a large vessel on the right side and two smaller ones on the left. The large vessel apparently corresponded to the innominate, and the other two vessels to the subclavian and common carotid of the left side. By these vessels blood was supplied to the head and upper extremities. There was no communication between the arterial and pulmonary vessels, as the ductus arteriosus was absent. The cause of the dilatation of the beginning of the aorta was a thickening and narrowing of the vessel for 8 mm. (⅓ inch) just beyond the dilatation. The heart was enlarged, but not especially hypertrophied.

There was a general streptococcus invasion, for which no source could be found. The cord had come away at the usual time without leaving any abnormal condition in the neighborhood of the umbilicus.

General Diagnosis.—Although it is usually possible to make a diagnosis of congenital cardiac disease, yet when we consider the variety of lesions which may occur, and the combination of different lesions which may be present, with the same symptoms or a marked lack of symptoms, it will be understood that a diagnosis of the especial lesion is often impossible.

Bearing in mind the mechanism of the fœtal circulation and the connection which an enlargement of the heart has with especial lesions, we can sometimes arrive at an approximately correct diagnosis. Too much reliance, however, must not be placed upon the locality or sound of the cardiac murmurs, as such murmurs may be produced by very trivial lesions, and may be absent when the lesions are most pronounced. The following characteristics may, however, be considered to represent the more common congenital anomalies.

OPEN FORAMEN OVALE.

One of the most common congenital malformations of the heart is represented by a defect in development of the auricular septum by an open foramen ovale. The failure of the foramen ovale to close is usually caused by an interference with the pulmonary circulation, such as may occur in atelectasis. Premature closure of the foramen ovale has been met with, but is extremely rare.

So long as the patent foramen ovale is not associated with other defects it does not, as a rule, produce a murmur or give rise to any symptoms. The reason for this is supposed to be that the blood can only flow from the right auricle into the left when the pressure is greater in the former, and it is not greater unless there is some obstruction either at the tricuspid valve or in the pulmonary valve or artery, by which the flow of the blood-current into the right ventricle is interfered with.

When symptoms such as cyanosis or murmurs are met with in connection with an open foramen ovale, they almost always depend on associated lesions, the most common of which are stenosis of the pulmonary artery and a defect in the ventricular septum. The prognosis in uncomplicated cases of patent foramen ovale is good, as the lesion is one which need not cause much embarrassment of the circulation.

DEFECT OF THE VENTRICULAR SEPTUM.

A defect in the ventricular septum is a very frequent congenital lesion. It may be complete, but is usually partial, and is most common in the upper part. This defect rarely occurs alone, but is associated with other lesions, especially stenosis of the pulmonary artery, and in such cases acts as a safety-valve. An abnormal origin of the great vessels is also frequently found with this defect. As the lesion is so frequently associated with other anomalies, there are no sharply marked and distinctive symptoms. In most cases, however, a loud systolic murmur is heard over the whole front of the chest, with its maximum intensity hardly ever near the apex, and with a notable absence of a palpable thrill. In uncomplicated cases hypertrophy of the ventricles is not marked.

When the right ventricle is strong enough to resist the increased blood-pressure from the left ventricle, the circulation is carried on normally without compensatory hypertrophy and without cyanosis or dropsy. When, however, great expiratory efforts occur, as in bronchitis and pertussis, or when the blood-pressure is increased from any other cause, dilatation of the right ventricle may set in acutely with symptoms of broken compensation, especially those of cyanosis and dropsy. These symptoms may, for a time, be delayed by a compensatory hypertrophy of the right ventricle.

The prognosis must necessarily be uncertain and unsatisfactory, as in so many instances the defective septum is accompanied by other lesions. In most instances the infants die early, but cases have been reported in which they have lived for many years.

LESIONS OF THE PULMONARY ORIFICE.

There are three lesions of the pulmonary orifice which play a very important rôle in congenital cardiac disease. These are stenosis or atresia of the pulmonary orifice and stenosis of the conus arteriosus of the right ventricle. These lesions may occur alone, but most commonly are associated with a defect in the septa and with a patent ductus arteriosus. They may be the result of a fault in development or of a fœtal endocarditis.

Pulmonary Stenosis.—This lesion is characterized by extreme cyanosis, cold, livid extremities, clubbing of the fingers, rapid respirations, and a tendency to dyspnœa. The physical, like the rational, signs are more characteristic than in the other forms of cardiac anomalies. They consist chiefly of a loud systolic murmur heard most distinctly in the pulmonary region and transmitted in all directions. There is, according to the extent of the stenosis, a weak or absent pulmonic second sound; if, however, the ductus arteriosus happens to be patent there may be no diminution in the intensity of the pulmonic second sound. In some cases a palpable systolic thrill may be felt in the pulmonary region. In many cases, however, the murmur obscures and dominates all other sounds. Generally there is an increased area of cardiac dulness extending to the right of the sternum and representing hypertrophy of the right ventricle.

Notwithstanding the apparent gravity of the lesion, pulmonary stenosis permits the infant to live, often for many years, but there is always in these cases a special tendency to tuberculosis and to pneumonia.

Pulmonary Atresia.—This lesion is less common but much more serious than stenosis. It is so commonly combined with other anomalies, such as defects in the septa and patent ductus arteriosus, that it cannot be said to have any distinctive physical signs of its own, while its general symptoms are the same as those of pulmonary stenosis.

Stenosis of the Conus Arteriosus.—This is an anomaly of development, is usually associated with other lesions of the orifice, and tends to accentuate the symptoms and results of such lesions. Pulmonary insufficiency has been observed in certain cases, but is very rare.

PERSISTENCE OF THE DUCTUS ARTERIOSUS

This lesion when existing alone may, as in a case which has come under my observation, present no symptoms except slight and evanescent cyanosis. The sign which, in addition to a light grade of cyanosis and a cadaverous color, is most characteristic of this condition is a loud vibratory systolic murmur, with its greatest intensity at the base of the heart. If the lesion is unassociated with other anomalies, there is no hypertrophy of the ventricles, but if, as so often occurs, there is obstruction at the pulmonary orifice, there is usually also hypertrophy of the right ventricle, and a diagnosis cannot be made, as the murmur cannot be distinguished from that of pulmonary stenosis.

The ductus arteriosus should gradually be obliterated within the first two weeks of extra-uterine life. Interference with this normal involution is not very uncommon, rarely occurs alone, and is usually found in connection with lesions of the pulmonary artery or narrowing of the isthmus aortæ. Sometimes the process of obliterative endarteritis extends to the aorta and causes stenosis of the isthmus aortæ. Again, the duct, in closing and retracting, pulls the aorta and tends to narrow that vessel, thus increasing the arterial tension. During fœtal life stenosis of the isthmus aortæ does not produce much disturbance in cases in which the ductus arteriosus can carry the blood to the descending aorta. At birth, however, in these cases, unless the ductus arteriosus remains pervious, serious symptoms arise. If life is prolonged, hypertrophy of the left ventricle takes place, and the arterial blood has to be conveyed to the descending aorta by means of a collateral circulation which is established between the branches of the subclavian arteries and the branches of the thoracic and abdominal arteries. Premature closure of the ductus arteriosus during fœtal life has been met with, but is a rare condition. Very rarely the ductus arteriosus may be entirely absent.

TRANSPOSITION OF THE LARGE ARTERIES.

A transposition of the aorta and pulmonary arteries sometimes occurs when the former arises from the right and the latter from the left ventricle. Both arteries may arise from a common trunk, and a number of other anomalies which are very rare may exist. Transposition of the arteries is not an uncommon anomaly, but there are no symptoms by which the condition can be diagnosticated, as there may be an entire absence of cyanosis, cardiac hypertrophy, and murmurs. The anomaly is, however, at times accompanied by intense cyanosis, asphyxia, cool skin and extremities, and apathy, but as it is always associated with other lesions, there are no distinctive symptoms by which a diagnosis can be made.

When this transposition of the main arterial trunks occurs the infant usually lives but a short time.

LESIONS OF THE TRICUSPID ORIFICE.

Tricuspid Stenosis.—This lesion is usually the result of a fœtal endocarditis. It is very rare and is often associated with deficient development of the right ventricle and hypertrophy and dilatation of the left ventricle.

Tricuspid Insufficiency.—This also is very rare, and results in hypertrophy of the right ventricle and dilatation of the right auricle.

Both of these lesions are accompanied usually by a number of other anomalies, so that there are no distinct symptoms by which to make a diagnosis, but, as a rule, the cyanosis is pronounced, the heart impulse is unusually strong, there are extensive systolic and diastolic murmurs, thrills, venous pulsation in the neck, epigastric pulsation, and a tendency to hemorrhage.

LESIONS OF THE MITRAL AND AORTIC ORIFICES.

Although lesions of the left side of the heart may be produced by fœtal endocarditis, they are exceedingly rare, in comparison with lesions of the right side of the heart, and are generally associated with a number of other congenital cardiac anomalies. Their symptomatic significance is the same as when they occur in extra-uterine endocarditis, and will be described under acquired diseases of the heart. It may be said, however, that the signs of tricuspid, mitral, and aortic lesions do not differ materially from those met with in adults.

The duration of life when the lesions are at the aortic orifice is not nearly so long as when the pulmonary orifice is affected.

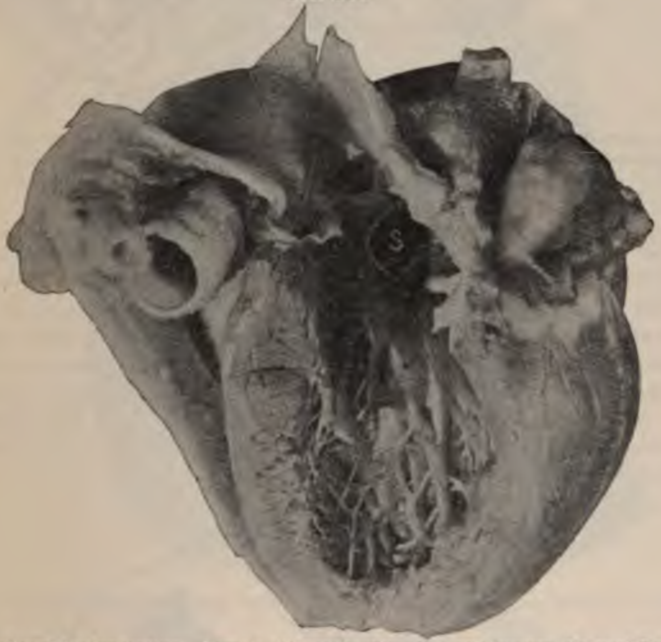
Differential Diagnosis of Congenital Disease of the Heart.—The differential diagnosis of congenital from acquired organic disease, and of the different varieties of congenital lesions, is exceedingly difficult at times, and often cannot be made. Certain facts, however, which seem to be generally accepted, may be stated, and will sometimes aid in the diagnosis.

Loud murmurs without much increase of cardiac dulness point towards congenital disease, while with increase of dulness they may be from either congenital or acquired disease.

If there are loud precordial murmurs without thrill and without hypertrophy, which are heard with greatest intensity about in the centre of the area of cardiac dulness, the lesion is probably a congenital defect of the ventricular septum.

If there is a thrill but no hypertrophy, the lesion is probably a congenital patent ductus arteriosus.

FIG 149.



S, unclosed ventricular septum. Female, 10 months old. Warren Museum, Harvard University.

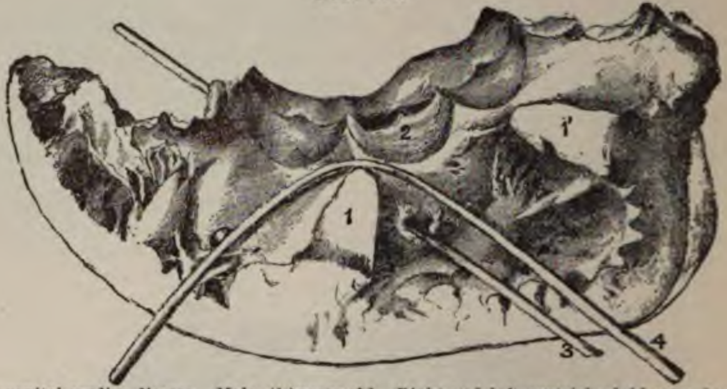
If there is a loud murmur at the base, with a weak pulmonic second sound, a thrill, and hypertrophy of the right ventricle, especially if there is marked and continued cyanosis, the lesion is probably pulmonary stenosis.

Murmurs of congenital origin must be distinguished from those of a functional or hæmic source. In congenital disease the murmurs are loud, often accompanied by a thrill, except in defect of the ventricular septum, are associated with cyanosis, dyspnœa, and cardiac hypertrophy, and are permanent. Functional murmurs, on the other hand, are seldom so loud, are evanescent, are unaccompanied by other marked signs, excepting often pronounced anæmia and a venous hum, and usually can be traced to definite etiological conditions such as are described on page 748.

Fig. 149 shows a small opening in the ventricular septum.

In this case there was also an open foramen ovale, but no other malformation. The infant, after showing the usual progressive signs of congenital cardiac disease, died suddenly. There was no history of cyanosis.

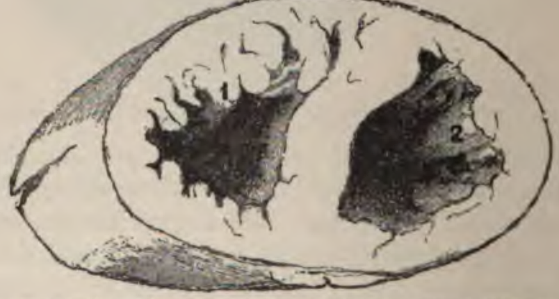
FIG. 150.



Congenital cardiac disease. Male, 4½ years old. Right and left ventricles laid open by two cuts. Stenosis of pulmonary orifice. Incomplete septum ventriculorum. 1 and 1', septum ventriculorum cut across; 2, aortic valves; 3, probe passing through narrowed pulmonary orifice; 4, bent probe, passing through right ventricle to left through opening in septum ventriculorum.

Fig. 150 represents a specimen taken from a boy four and a half years old, who during life had shown cyanosis, clubbed fingers, and at times severe dyspnœa. The physical signs in connection with the heart were a fine wave perceptible to the eye at the left third interspace, a soft, purring thrill over the base of the heart, cardiac pulsation 1.4 cm. (½ inch) outside of the left mammillary line, and cardiac dulness from the right sternal margin to the left mammillary line, with no dulness to the right of the sternum. A loud, harsh systolic murmur was heard over the left margin of the sternum, most marked at the second left interspace and third rib, and not transmitted to the left or along the aorta.

FIG. 151.



Transverse section of heart near apex. 1, right ventricle; 2, left ventricle.

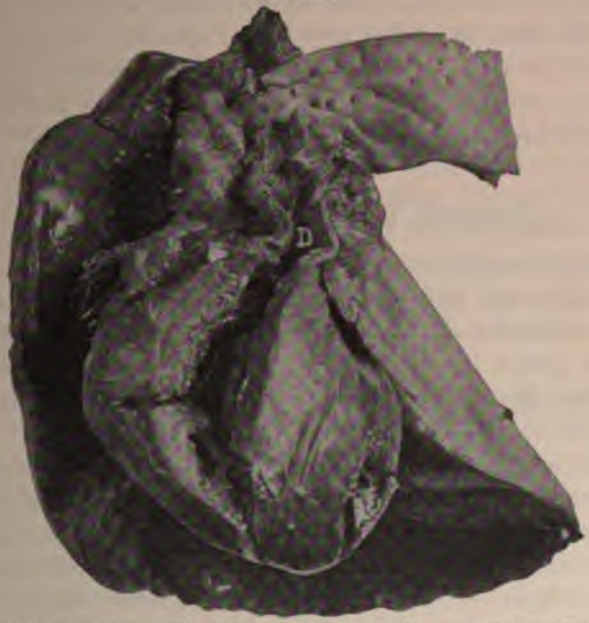
The pulmonary artery was abnormally small, the aorta was abnormally large, the conus arteriosus was practically obliterated at the pulmonary orifice, and the ventricular side formed a ring of white cicatricial tissue ½ cm. (⅕ inch) in diameter.

Fig. 151 represents the same heart with the apex cut away so as to show the relative thickness of the ventricular walls and the greatly thickened septum ventriculorum.

The right ventricle was markedly hypertrophied. The left ventricle was normal. The ventricular septum was greatly hypertrophied. In this case the ductus arteriosus was impervious and the foramen ovale practically closed. A fœtal endocarditis had taken place before the septum ventriculorum had closed. The endocarditis caused contraction of the conus, and the blood being forced from the right ventricle through the imperfect septum prevented the latter from closing. This provided a safety-valve, which, as usually happens in this form of malformation, allowed the child to live longer than is common in other congenital cardiac malformations. The aorta, receiving a direct stream from both ventricles, was distended; the pulmonary artery, receiving but little, remained small. It is interesting to note in this case that the child passed through an attack of pertussis and measles without serious results. It died ultimately of abscess of the brain.

Fig. 152 shows an open ductus arteriosus.

FIG. 152.



D, open ductus arteriosus. Male, 16 days old. Warren Museum, Harvard University.

This heart, which was left attached to the lung, was taken from an infant, sixteen days old, who was apparently healthy at birth and presented no symptoms of cardiac disease.

When the infant was five days old it was noticed that it would sometimes become slightly cyanotic. At this time its temperature rose to 39.4° C. (103° F.). A physical examination showed nothing abnormal, and nothing unusual was seen on inspection. The area of cardiac dulness was normal, and no murmurs were detected. A day or two later the temperature became normal; the cyanosis increased somewhat, but was intermittent and of a very slight degree. At times the skin would become cool. A few days later there was slight intestinal disturbance. When sixteen days old, without any other symptoms having developed, the infant died suddenly. The post-mortem examination showed this widely open ductus arteriosus. The foramen ovale was also open. There were no other lesions, such as stenosis of the pulmonary artery, open ventricular septum, or lesions of the valves. The heart was of normal size. There were no signs of the obliterative endocarditis usually found at this age in the ductus arteriosus.

General Treatment.—The treatment of congenital disease of the heart is essentially hygienic and symptomatic. The infants should be carefully protected from atmospheric changes which would be likely to produce bronchial irritation, as in many cases bronchitis appears to play an important part in interfering with the maintenance of the equilibrium of the circulation and in destroying compensation. In a number of cases I have found that the administration of digitalis in small doses and with the greatest caution is valuable when hypertrophy has begun to fail and dilatation to increase. When the dyspnœa is distressing, a few drops of aromatic spirits of ammonia will often give relief. Stimulants are usually indicated.

Freedom from excitement and over-exertion should be constantly enforced, but the child should be kept in the open air, when it is warm and dry, as much as possible. Finally, infants and children with congenital cardiac disease should be closely watched, even when they seemingly are doing well, for they are apt to die suddenly.

ACQUIRED DISEASES OF THE HEART.

In studying acquired diseases of the heart it is important to recognize the relative size and position of the heart at different ages. These have been described on page 90. Acquired diseases of the heart are more apt to attack the left side of the heart than the right; when the right heart is affected it is generally secondary to the lesions on the left side.

There are certain differences between the symptoms of cardiac disease in infancy and early life and those in later life. In young children murmurs are more apt to be diffuse than in adults, often being heard over the entire chest; and the rate and rhythm of the heart are so easily disturbed by nervous influences as to be of little diagnostic value. Progressive emaciation is a symptom which is apt to be pronounced and to appear early in the disease, especially if the child is young.

An enlarged heart dependent on adhesions from a preceding pericarditis is more common in early life than in adults, while compensation is much more readily acquired. I have had children with cardiac disease at my children's clinic one year with cardiac symptoms so severe that they had to be carried; they were emaciated and cyanotic, the area of cardiac dulness was increased, and souffles were present; yet these same children would return and be shown to the next class of students in the following year, walking up-stairs without dyspnœa, looking well nourished, of good color, with much less enlargement of the area of cardiac dulness, and with the cardiac souffles scarcely perceptible, showing that the cardiac compensation was complete.

Cardiac symptoms dependent on organic lesions may arise and yet no physical signs of such lesions be detected during life.

CARDIAC HYPERTROPHY.

Hypertrophy of the heart consists of an increase in thickness of the cardiac walls. It may be of one or both sides of the heart and is usually most marked in the ventricles. The hypertrophy may be of the walls without an accompanying dilatation of the cardiac cavities (eccentric), or dilatation of the cavities may be present, thus increasing the entire size of the heart. Cardiac hypertrophy is always an effort of nature to counteract various morbid influences by maintaining the equilibrium of the circulation,—that is, it is compensatory.

Etiology.—Hypertrophy of the heart is not in itself a disease, but may occur in many diseases whether of the walls or of the valves. It is closely related to increased blood-pressure, which may arise from disease or circulatory disturbance outside of the heart or from imperfect valvular conditions within the heart.

In the former case the most common causes in children are pericardial adhesions, prolonged pertussis, narrowing of the aorta, and nephritis, especially that which follows scarlet fever. Mechanical interference with the action of the heart arising from adhesions of the pericardium is often latent in its symptoms in infancy, and its occurrence should be especially borne in mind. In the valvular lesions the increased demand for cardiac work beyond what is normally called for acts as an immediate cause for cardiac hypertrophy.

Pathology.—The chief morbid conditions of cardiac hypertrophy are an increase in weight and an increase in size and number of the muscular fibres which is found not only in the cardiac walls but in the papillæ and trabeculæ. If the left side of the heart is conspicuously enlarged, as in obstruction at the aortic orifice or in the general arterial system, the heart is elongated; if the hypertrophy is of the right side, as in obstruction of the pulmonary circulation, the heart is widened.

Symptoms.—So long as the compensation is complete, cardiac hypertrophy does not produce in young subjects any marked symptoms, even the dyspnœa on exertion not being especially noticeable.

Hypertrophy of the Left Ventricle.—In hypertrophy of the left ventricle the cardiac impulse is perceptible over an abnormally large area and the impulse is lower and further to the left than normal, the degree varying according to the extent of the hypertrophy.

Palpation shows the impulse to be increased in force. Percussion shows the cardiac dulness to be markedly increased in extent downward and to the left. Auscultation shows the first sound at the apex to be long and low in comparison with the second sound, which is loud and sharp. There is accentuation of the aortic second sound. The pulse is full and strong.

Hypertrophy of the Right Ventricle.—In hypertrophy of the right ventricle the cardiac impulse is especially marked further to the left than normal and somewhat downward. There is nothing characteristic in the pulse. The area of superficial cardiac dulness is increased in width, notably to the right and often beyond the right sternal line. There is accentuation of the pulmonic second sound, but this accentuation, although almost always present in hypertrophy of the right ventricle, is not peculiar to this condition, as it may be present in varying degrees in young children normally and from such causes as pneumonia and certain nervous conditions.

Prognosis.—So long as full compensation is present the prognosis is favorable. It becomes proportionately unfavorable as the compensation lessens.

Treatment.—The treatment is described on page 746.

CARDIAC DILATATION.

Cardiac dilatation occurs when the cavities of the heart are abnormally increased in volume. In many cases there is a thinning of the walls of the heart, as in acute dilatation, but when hypertrophy is present, as in certain chronic cases, the walls may be thicker than normal.

Etiology.—*Acute dilatation* may occur from a weakening of the cardiac muscle caused by degeneration of the myocardium, as in certain infectious diseases and from poor nutrition.

Chronic dilatation occurs slowly as the result of valvular lesions or in the progress of the circulatory disturbances spoken of as causing hypertrophy.

Pathology.—In simple dilatation, without hypertrophy, the weight of the heart is not increased, although there is increase in its size. The papillæ are flattened, when the dilatation is marked, in comparison with the accompanying hypertrophy, and the endocardium is thickened and opaque.

Symptoms.—The symptoms of cardiac dilatation are essentially those of a failure of compensation characterized by dyspnœa on exertion, palpitation, cough, and, later, cyanosis and œdema. In both acute and chronic dilatation there are a weak, fluttering apex-beat and pulse, an increased area of cardiac dulness, and valvular murmurs varying according to the especial lesions.

Dilatation of the Left Ventricle.—There is an increased area of cardiac dulness to the left and downward. The first heart-sounds are sharp and short. The aortic second sound is feeble both at the apex and in the aortic area.

Dilatation of the Right Ventricle.—The chief signs of dilatation of the right ventricle are increase of the area of cardiac dulness to the right, corresponding to the region of the right auricle, and a weak pulmonic second sound.

In all the conditions which cause dilatation there is a greater liability that acute dilatation may take place in early life than at a later period.

The processes which suddenly cause great increase of the blood-pressure in the lungs may lead to acute dilatation of the right ventricle. When there is a diffuse renal disease, such as may develop in the course of scarlet fever, acute dilatation of the left ventricle may take place, and be followed by hypertrophy. In all these diseases this acute dilatation may take place rapidly and disappear almost as rapidly, a phenomenon which is somewhat characteristic of cardiac disease in early life.

The great changes which take place in the heart and its rapid growth at the time of puberty have already been referred to. At this period the general growth of the child is apt to be very rapid, and symptoms of cardiac weakness commonly occur, especially in girls. These symptoms are debility, lack of energy, palpitation, and dyspnœa on exertion. There may also be signs of slight cardiac dilatation, and murmurs, probably hæmic in their nature. This period, therefore, is one in which cardiac disease from any cause, such as rheumatism, is of more serious import than at a later period, when the heart is not taxed by too rapid growth.

PROGNOSIS.—The prognosis of cardiac dilatation depends on whether the cause is a temporary one or whether, in case it is not, compensatory hypertrophy can be obtained by treatment. Whether the prognosis is favorable or unfavorable must, then, be determined by the evidence of reaction which is presented by the individual case.

TREATMENT.—The treatment is described on page 746.

MYOCARDITIS.

Primary disease of the myocardium is very rare in children, in whom disease of the myocardium is almost always a secondary process resulting from one of the infectious diseases. It may be acute or chronic.

ETIOLOGY.—The most common causes in children are endocarditis, pericarditis, and the infectious diseases, especially scarlet fever and diphtheria. The other causes which may produce an affection of the myocardium in later life may occur at any age, but are too rare in children to be considered. They are found in lesions of the coronary arteries (anæmic necrosis), an excess of food, alcohol, physical strain, and arteriosclerosis.

PATHOLOGY.—The lesions may be parenchymatous or interstitial, degenerative or inflammatory. They are essentially of the septum and left ventricle. In the *parenchymatous form* the anatomical changes are in the muscle-cells, the cells being degenerated in various ways (granular, fatty, hyaline), and the muscular fibres often separated, a condition known as fragmentation and segmentation. This form is usually met with in fevers, intoxications, infectious conditions, and in connection with endocarditis and pericarditis. It is always acute, usually diffuse, and is especially characterized by softness of the heart.

The *interstitial form* may be acute or chronic, and occurs in small areas. In the acute variety, such as occurs in certain infectious diseases, most commonly in diphtheria and typhoid fever, and especially in acute pericarditis, which is an important local cause in childhood, there is an infiltration of round cells between the fibres, and in some cases small abscesses are formed. In the chronic variety there are opaque, white or yellow spots which later may soften (Ziegler's myomalacia), and, as has occurred in rare cases in children, cardiac aneurism and rupture may result. The parenchymatous and interstitial lesions of the myocardium may develop alone or in combination. The other pathological lesions of the myocardium are so rare in children that they need merely be mentioned. They are *fatty degeneration* and *infiltration*, *brown atrophy*, *amyloid degeneration*, *hyaline transformation* (Zenker), and *calcareous degeneration*.

SYMPTOMS.—The symptoms are essentially those of a weak heart, but as to whether such symptoms are indicative of an affection of the myocardium, or of nervous conditions of the heart, or of some other cardiac disease is usually very uncertain.

In acute cases there may be palpitation, pallor, dyspnœa on slight exertion, a rapid, irregular, weak pulse, disturbance of digestion, and on auscultation feeble heart-sounds, especially the aortic second, and in the beginning absence of murmurs. Usually there is no increase in cardiac dulness.

It must, however, be remembered that these same symptoms may be present and yet no lesion of the myocardium be found at the autopsy, while, on the other hand, without any especial symptoms referable to the heart, sudden death may occur and marked lesions of the myocardium be present.

In chronic cases the weakening of the heart is slow and progressive and the area of cardiac dulness is increased, corresponding usually to the left side of the heart. The impulse is feeble and murmurs may eventually appear as the valves of the heart become incompetent. Symptoms of venous stasis gradually supervene. The pulse is irregular and small; it is sometimes slow, but becomes rapid under even slight nervous excitement. All these symptoms may become intensified, but it must be borne in mind that other diseases may cause these same cardiac symptoms and that the autopsy may disclose serious chronic lesions of the cardiac walls where during life no cardiac symptoms had appeared.

DIAGNOSIS.—The diagnosis depends upon symptoms of cardiac weakness, with a rapid, irregular, feeble pulse, without murmurs, with or without increased cardiac dulness, upon the progressive character of the symptoms, and upon the comparatively slight improvement under treatment. The etiological factor in the diagnosis is important. When a previously healthy child has been attacked by one of the infectious diseases, such as scarlet fever, diphtheria, or typhoid, and when cardiac symptoms not referable to an endocarditis or pericarditis arise, we may suspect a lesion of the myocardium.

PROGNOSIS.—The prognosis in affections of the myocardium depends upon the cause of the condition and also, if it is secondary to one of the infectious diseases, upon the degree and virulence of the infection. It is, therefore, very uncertain in the acute cases, although always serious, while in the chronic cases it is very grave.

TREATMENT.—The treatment is essentially rest and freedom from nervous excitement and sudden exertion, as from sitting up in bed. After such diseases as diphtheria a recumbent position is often indicated for weeks when marked signs of cardiac weakness have arisen; and this may be said also when these symptoms supervene on any of the infectious diseases. Stimulants such as aromatic spirits of ammonia and alcohol should be freely used. Strychnine is, of all drugs, perhaps the most valuable, while digitalis, if used at all, should be given with great caution and in small doses. When there is much precordial distress and the symptoms of cardiac failure are imminent, small doses of morphia given subcutaneously are indicated. Anæmia and other symptoms, if present, should receive appropriate symptomatic treatment.

ENDOCARDITIS.

Endocarditis is an inflammation of the endocardium which is usually confined to the valves. It may be acute or chronic.

Acute Endocarditis.—The acute form of endocarditis may occur in fœtal life, and in these cases almost invariably affects the right side of the heart, while if the disease is acquired in extra-uterine life it is the left side of the heart which is most frequently attacked.

ETIOLOGY.—Acute endocarditis may, in rare instances, be a primary disease, but in most cases is caused by some one of various infectious processes. In some cases bacteria have been found, but not in all. There is no one variety of bacteria exclusively concerned in the production of acute endocarditis. The streptococcus, staphylococcus, pneumococcus, and gonococcus are the forms which have been most frequently found. Less frequently, especially in the simple form of acute endocarditis, the tubercle bacillus, the typhoid bacillus, and the anthrax bacillus have been met with. The colon bacillus, diphtheria bacillus, bacillus pyocyaneus, and bacillus of influenza have also been reported. There is no difference in the endocarditis arising from these various organisms except in the degree of the malignant nature of the especial organism which has produced the disease, or in the vulnerability to infection of the individual.

The frequent occurrence of acute endocarditis in children has been attributed to the greater vulnerability to bacterial action in early life.

Acute endocarditis may be of the *simple* or *verrucose* or of the *septic* or *malignant* form; this distinction, however, is based on the intensity of the endocarditis rather than on marked etiological differences. The malignant form is very rare in childhood.

The most frequent etiological factor in simple acute endocarditis is acute articular rheumatism. It may also occur in chorea either with or without a rheumatic complication. While it may occur in the course of any of the infectious diseases, such as influenza and diphtheria, it is most common in scarlet fever, pneumonia, and pleurisy, while it is not common in measles and varicella. The malignant form of endocarditis seems to occur more frequently in pneumonia than in any of the other acute infectious diseases.

PATHOLOGY.—While the same lesions of endocarditis may be found in children as in adults, yet in infancy, although marked acute cardiac symptoms and murmurs frequently arise, the autopsy almost invariably fails to show any endocardial lesions or growths. In two thousand autopsies at the New York Foundling Asylum, Dr. Northrup and Dr. O'Dwyer never found an acute inflammatory lesion, except in one case, which showed the lesions of acute malignant endocarditis. When the lesions of endocarditis are found in children, the connective tissue and the basement substance are principally concerned in the inflammatory process. The endocardium which forms the valves is that which is most frequently inflamed, but other portions of it are by no means exempt. In some cases there is swelling of the valves, which are thickened, their surfaces remaining smooth, the basement substance is swollen, and there is a moderate production of new connective-tissue cells. In other cases the growth of connective-tissue cells is very much more marked, the basement substance is broken up, and little cellular fungus-masses, called vegetations, project from the free surface of the endocardium (verrucous endocarditis).

On the surface of these vegetations thrombi may be formed, and bacteria are sometimes present. In still other cases the cellular growth in some places forms vegetations, and in others degenerates, and thus portions of the valves are destroyed. This is *acute ulcerative endocarditis*, also called *malignant endocarditis*. In these cases the diseased valve and the thrombi contain bacteria much more frequently and in far greater numbers than are found in the simple benign form. This malignant form is evidently secondary in the course of a septic infection, and the previous disease of the valves favors the invasion of the infectious bacteria. In the simple form of acute endocarditis emboli may occur, which from their size usually produce mechanical disturbances alone, while in the malignant form the emboli are usually smaller, and, being septic, produce inflammatory rather than mechanical disturbance, giving rise to miliary abscesses in various parts of the body, such as the liver, kidneys, spleen, stomach, intestine, brain, eye, joints, and skin. Acute endocarditis may also be secondary to old cardiac malformations or lesions (endocarditis recurrens).

In some cases the children recover, and the valves seem to return to their normal condition, while in others the valves are left permanently damaged.

Simple Acute Endocarditis.—SYMPTOMS.—The symptoms of simple acute endocarditis are often obscure, and in infants and young children in the beginning are apt to be latent. When the disease arises in connection with some other disease, the symptoms are especially likely to be masked by those of the disease which it complicates. In some cases the endocarditis develops insidiously without any additional symptoms, such as pain or precordial distress, and its presence is not recognized until a careful examination of the heart detects a murmur; in others, pronounced and even violent cardiac symptoms are present from the beginning. Again, the first evidence of an endocarditis may come from symptoms of embolism such as hemiplegia, hæmaturia, dyspnœa, cough, or localized pain, as in the thorax or abdomen. If the muscular tissue is involved as well as the endocardium, the general cardiac symptoms of dyspnœa, cyanosis, and palpitation are still more marked.

When endocarditis arises in the course of acute rheumatism there may be nothing which would draw especial attention to the heart, but there are usually general symptoms of increased rapidity of the heart's action, with perhaps slight irregularity, restlessness, prostration, and a rise in the temperature, which is especially suspicious if, with this increase in the fever, there is no exacerbation of the trouble in the joints. Palpitation and dyspnœa are also quite frequently present. There may at first be no murmur, but usually after a few days a soft systolic murmur at the apex transmitted to the left, and later a slightly accentuated pulmonic second sound, may be heard. Endocarditis may with these same symptoms occur in the course of chorea.

When endocarditis does not arise as a complication of some other disease, the symptoms at the onset, when prominent, are usually a rise of temperature, an accelerated and sometimes weak and irregular pulse, dyspnœa, palpitation, and more or less precordial distress. All these symptoms vary according to the extent of the lesions. Later they depend upon whether or not compensation has been established. In connection with these early symptoms, cardiac dilatation and cyanosis are very apt to occur. When the disease has advanced so far as to interfere with compensation, the physical signs of dilatation appear. The symptoms differ somewhat according as the inflammatory condition has begun in the valves or in the cardiac walls (Steffen). In the former case the signs of dilatation accompany those of valvular weakness, while in the latter the symptoms of dilatation come first, and are followed by the mechanical results of valvular insufficiency.

In a first attack of acute endocarditis the serious symptoms connected with great lack of compensation which are met with when the attack supervenes on a previous cardiac lesion are not likely to arise. In some cases, however, when the individual power of cardiac resistance is slight, they appear early in the disease. Under these circumstances the child emaciates rapidly, becomes very weak and anæmic, and the cyanosis and dyspnœa increase. There is apt to be cough from an accompanying bronchial irritation, produced most frequently when there is obstruction at the mitral orifice. As a result of a general venous stasis, enlargement of the liver, hæmoptysis, and œdema of the face, legs, and arms appear. Children show such a wonderful recuperative power that even in these advanced cases under proper treatment the serious symptoms may gradually pass away, and often such complete cardiac compensation takes place that they are left with no symptoms of cardiac disease except a murmur and some hypertrophy.

Relapses are common and there is a great tendency to recurrence. Embolism may take place as a late as well as an early complication. Anæmia is a very common symptom, especially when endocarditis accompanies rheumatism. Congestion of the lungs with resulting hæmoptysis may arise when there is insufficiency of the mitral valve. When the valves are affected murmurs are usually present, yet sometimes when there are lesions of the valves murmurs cannot be detected. In endocarditis murmurs are most frequently heard in the region of the mitral valve, and insufficiency of the mitral valve is the most common of the inflammatory cardiac lesions in childhood.

Acute simple endocarditis may last for several weeks, the symptoms gradually subsiding or becoming chronic; or there may be recurrent attacks which are especially likely to result in chronic valvular disease; or acute dilatation may result with increasing symptoms of cardiac insufficiency, dropsy, and pulmonary complications, finally ending in death.

DIAGNOSIS.—The diagnosis of simple acute endocarditis during life depends upon the physical signs. These signs are an increase in the area of cardiac dulness and a change in the sounds of the heart. The change in the area of cardiac dulness must be differentiated from that which occurs in a pericardial effusion as described on page 759.

The change in the cardiac sounds may be produced by changes in the blood or by organic lesions of the valves. The differential diagnosis between these two conditions is given on page 748, and the diagnosis of the especial valvular lesion is given on pages 741–745.

PROGNOSIS.—The prognosis of simple acute endocarditis in early life is comparatively favorable as to the immediate outcome, but may eventually become serious by the occurrence of embolism or the development of a malignant endocarditis. Owing to the great recuperative powers in early life, in many cases, especially when it is the first attack, such complete compensation takes place that the child practically recovers. If it is the walls of the heart that are affected, the heart may regain its normal size and position. If the valves alone, or the valves and the walls, are affected, recovery can still take place. The development of chronic valvular disease is the usual termination. Death may, however, occur at the height of the attack, or the child may die later from exhaustion and sometimes suddenly from heart-failure.

Malignant Endocarditis.—SYMPTOMS.—The symptoms of malignant endocarditis are necessarily varied and indefinite, arising as they usually do in the course of some other disease with simply an exacerbation of the previous symptoms, especially of the temperature. There are usually the general symptoms of intermittent temperature, sweating, delirium, and failure in strength. Two types of the disease, septic or pyæmic and typhoidal, are recognized. The *septic type* is characterized by the signs of septic infection, rigors, sweating, and irregular temperature, while the *typhoidal type* shows diarrhœa, dry tongue, distended and tympanitic abdomen, enlargement of the spleen, profuse sweating, and a petechial efflorescence on the skin. A clear distinction between the two forms, however, is often not marked. Cerebral symptoms may arise, and simulate meningitis and malaria. Embolic processes may produce special symptoms, such as paralysis, coma, bloody urine, retinal hemorrhage, peritonitis from infarction of the spleen, and in some cases pulmonary symptoms simulating pneumonia. Murmurs may be absent. The duration of the attack depends mostly on the disease to which it is secondary, and may vary from a few days to as many weeks. A marked leucocytosis is present.

Diagnosis.—The diagnosis of malignant endocarditis is made from the simple form by the graver constitutional signs, the higher temperature, and the septic or typhoidal symptoms just described. From typhoid fever it is to be distinguished by its abrupt onset, irregular temperature, precordial pain and distress, marked leucocytosis, and negative Widal reaction. Malaria can be eliminated by the absence of the plasmodium malariæ and the presence of leucocytosis.

Prognosis.—The prognosis of malignant endocarditis is very grave, although the disease is not necessarily fatal. The heart is, however, permanently damaged.

Treatment of Acute Endocarditis.—The treatment of acute endocarditis during the early days of the attack is essentially rest in bed. When the disease is due to some specific disease, such as diphtheria, the specific remedy for that disease should be given at once. From the very beginning, however, we should endeavor to establish compensation. The child should be encouraged to sleep, in order that the circulation may be kept as quiet as possible and thus relieve the work of the disabled heart. The heart-beats of a young child during sleep are often reduced twenty in a minute, and thus sleep affords the best opportunity for compensation. Later the general health of the child should be carefully attended to by means of good food, pure air, and exercise of a mild type, never excessive. The surface circulation should be promoted by baths and gentle massage. Digitalis and iron are of great value, the former in aiding the establishment of compensation, the latter in combating the anæmia. When there are symptoms of heart-failure, stimulants such as alcohol, aromatic ammonia, and strychnine (page 470) are indicated and important. If at any time during the course of the disease the attacks of dyspnœa are excessive, nitroglycerin can be given in doses proportionate to the age of the child; 0.0003 gramme ($\frac{1}{200}$ grain) can be given to a child three or four years old. The younger the child, the more likely it is that we shall have to contend with a resulting atrophic condition and anæmia.

The treatment of the malignant form of endocarditis is essentially that of a septic condition, and stimulants should be used freely.

CHRONIC ENDOCARDITIS.

Chronic endocarditis showing sclerosis of the valve may be chronic from the outset, but it usually succeeds an acute endocarditis; this occurs especially in the rheumatic form.

ETIOLOGY.—The usual causes of chronic endocarditis in later life, such as syphilis, alcohol, gout, and strain from undue muscular exertion, are very seldom met with in children, acute endocarditis being the chief etiological condition.

PATHOLOGY.—Although the morbid process may attack the parietal endocardium, it is the valvular form that is of such clinical importance as to become the prominent feature in chronic endocarditis. The process in extra-uterine life most frequently affects the aortic and mitral valves, the latter being especially involved in childhood. It is a slow, insidious process, manifested by thickening, induration, and adhesions, followed by a contraction of the valvular segments. Later, lime-salts are deposited in the valves and this results in rigidity. As the sclerotic changes increase, the deformity of the segments becomes greater and results either in imperfect closure of the valves (*valvular insufficiency*), allowing a regurgitation of the blood, or the orifice is narrowed (*valvular stenosis*), causing obstruction to the blood-current. We meet, especially in children, with lesions at the mitral orifice in which the valves are curled and thickened so that they are insufficient, although there may be no narrowing of the orifice. The chordæ tendineæ gradually become shortened. The apices of the papillary muscles are enlarged, the endocardium becomes thickened, and marked changes following the valvular lesions take place in the walls of the heart. These changes are represented by varying degrees of hypertrophy or dilatation according to the degree and duration of the valvular lesions and to the extent to which compensation has been interfered with. In the order of their frequency the mitral valve is most often affected, next the aortic, then the tricuspid, and very rarely the pulmonary valve. It is not uncommon for the aortic and mitral valves to be affected together. In very rare cases all the valves may be involved.

GENERAL SYMPTOMS.—The symptoms of chronic endocarditis develop gradually, especially when compensatory hypertrophy is complete. They often extend over a period of many months, corresponding to the slow progressive nature of the valvular lesions. When there is a leakage through the valves or when there is obstruction to the flow of blood an increased amount of work is thrown upon the auricles and ventricles, and symptoms of hypertrophy or dilatation result which are directly in proportion to the amount of compensation which is present. The final effect, therefore, of all valvular lesions is a failing compensation and venous stasis throughout the whole body.

In children there may be long periods when the morbid conditions do not increase and when, beyond occasional attacks of dyspnœa on exertion, other symptoms are not prominent.

Symptoms, such as cough, epistaxis, palpitation, and anæmia, are apt to appear and then pass away, when for any reason the child is debilitated. A decided failure in compensation is liable to arise at any time from an attack of acute endocarditis, from over-exertion, or from the presence of one of the infectious diseases, such as scarlet fever or typhoid.

As compensation fails the corresponding symptoms of a weak heart become more marked. There are present some, rarely all, in young children, of the following symptoms: increased dyspnœa, orthopnœa, bronchitis, dropsy, beginning in the feet, enlarged liver and spleen, ascites, renal congestion with albuminuria, indigestion, and, later, prominence of the superficial veins, clubbed fingers, and cyanosis. Emaciation is a marked symptom. There may be at any time during the course of the disease cerebral symptoms such as fainting and dizziness, and sometimes headache. These symptoms, even when extreme, may all pass away, and then at varying periods of months or years return. Death may take place from sudden paralysis of the heart or as a result of the additional tax on the heart from some intercurrent disease, such as pneumonia or nephritis. Embolism, with its resulting paralysis or œdema of the lungs, may hasten the fatal result.

In connection with these symptoms certain physical signs of the heart itself are present which correspond to the especial valvular lesions which are present.

Mitral Insufficiency.—Incompetency of the mitral valve with regurgitation is the most common of all the valvular lesions, and in most cases is caused by rheumatism. It is also especially met with in chorea. Before compensation has taken place, the lesion is represented by a systolic murmur most marked at the apex, synchronous with the impulse and with the first sound, which it may replace, and transmitted into the axilla and back. When compensation has taken place there is an accentuated pulmonic second sound and an increased area of cardiac dulness, corresponding at first to enlargement of the left auricle and right ventricle and later of the whole heart. In this latter condition there is not only an increase in the breadth of the heart, but also in its length, the apex being depressed and carried in varied degrees to the left of the mammillary line. There may be bulging of the precordia. The aortic second sound is diminished. The pulse is diminished in volume and tension.

As compensation fails the general symptoms of a weak heart appear, and dilatation predominates over the hypertrophy with the sequence of symptoms already described.

Mitral Stenosis.—Stenosis of the mitral valve is usually the result of chronic endocarditis. It develops slowly and is often a later stage of the same process which has earlier produced mitral regurgitation. It occurs most commonly in girls, and many cases are associated with tuberculosis of the lungs.

In the early part of the disease there is little or no increase in the cardiac dulness, but later the cardiac area is increased in breadth and corresponds to an enlarged left auricle and right ventricle. A sign which may be said to be characteristic of mitral stenosis is a thrill, which on palpation gives rise to a purring sensation, felt at the apex of the heart, just before and up to the time of the systole, when it ceases sharply. As in mitral regurgitation, there may be bulging in the precordial region, especially to the left of the sternum and within the mammillary line. The impulse is both seen and felt rather higher in the vertical line than is normal, and is due to the impulse of the conus arteriosus of the right ventricle. A presystolic murmur, rough and purring in quality, is heard in the mitral area over a rather limited space, and is not markedly transmitted. It begins shortly after the end of diastole and increases with a crescendo until systole begins, when it ceases abruptly with a very sharp first sound. The thrill can sometimes be felt when no murmur is heard, and can be transmitted into the axilla, while the murmur is only heard in the mitral area; in some cases, however, the murmur is transmitted into the axilla. The second sound is frequently reduplicated, and this is an important diagnostic sign, as it may be heard in some cases in which the murmur is absent. Irregularity in force and rhythm may occur at any stage.

The pulmonic second sound is accentuated, may be reduplicated, and may be transmitted a considerable distance to the left. In uncomplicated cases there is usually no murmur heard in the aortic area. In the later stages of the disease, corresponding to marked dilatation of the right ventricle, tricuspid regurgitation develops and the murmur may disappear. The sharp first sound remains, and, with prolongation of diastole and great cardiac irregularity, is of much aid in the diagnosis at this stage. When in conjunction with a reduplicated second sound, it suggests mitral stenosis, which may be masked by the prominence of the signs of mitral regurgitation, which is so commonly associated with mitral stenosis.

It is rather characteristic of mitral stenosis that, although the pulse may be markedly irregular, the general circulation remains good for long periods. Embolism is a very common complication, even more so than in lesions of the other valves. Hæmoptysis is also apt to occur.

Finally, compensation fails and the symptoms are the same as in mitral insufficiency with cardiac dilatation. Dropsy in many cases is absent. Hepatic enlargement and ascites are common. The increased area of cardiac dulness is often masked by the increased size of the lungs.

The prognosis of pulmonary and pleural affections arising in the course of a mitral stenosis is especially bad.

A presystolic murmur at the apex may be heard in some cases of aortic regurgitation, but the murmur may be considered to arise from mitral stenosis if with absence of a predominating hypertrophy of the left ventricle there are the signs of pulmonary congestion, accentuation of the pulmonic second sound, and a sharp mitral first sound. Tricuspid obstruction is almost impossible to differentiate from mitral stenosis, but is exceedingly rare. Following an attack of pericarditis a murmur may be heard which closely simulates mitral stenosis, but is differentiated by absence of the sharp first sound at the apex and the other signs of pericarditis.

Aortic Insufficiency.—Lesions of the aortic orifice are rare in childhood, as rheumatism, the most frequent cause of endocarditis in children, in almost all cases attacks the mitral valves. Aortic disease may therefore be said to belong essentially to a later period of life, and, when it does occur in early life, to be associated almost invariably with some other valvular lesion, usually the mitral. In most cases there is a thickening and shortening of the cusps, causing a regurgitation of the blood from the aorta into the left ventricle during diastole. As a result of the increased amount of work thrown on the left ventricle dilatation and hypertrophy occur, producing greater ventricular enlargement than any other form of valvular disease. As a result there is marked heaving, throbbing, and often bulging in the precordia. There is great increase of the cardiac dulness, especially to the left and downward.

Aortic insufficiency is shown by a rather faint murmur coincident with or replacing the second aortic sound. The maximum intensity of this murmur seems to vary in different cases. It may be loudest in the aortic area, but is most common in the midsternal region, especially on the left side of the sternum at about the level of the fourth left costal cartilage. The murmur in some cases is also heard distinctly near the apex of the heart, and less frequently its maximum intensity may be at the second or third left costal cartilage or over the ensiform cartilage.

Although in a certain number of cases the murmur may be transmitted into the axilla and even into the back, as a rule the transmission is limited to the precordia, although it may extend in all directions, but especially downward towards the apex. The aortic sounds, and sometimes the murmur, may be transmitted into the carotid and subclavian arteries. The carotid arteries may show evident pulsation, and there is the characteristic Corrigan pulse. The first sound of the heart at the apex is prolonged. The pulmonic second sound is not accentuated. The general symptoms usually come on slowly, and are characterized at first by dyspnœa, headache, and other signs of cerebral congestion.

Compensation may last for long periods, but when it fails symptoms of faintness, precordial pain, angina pectoris, and sometimes, although rarely, dropsy and cyanosis appear, these latter two symptoms usually occurring when an associated relative insufficiency of the mitral orifice has taken place. Death follows quite frequently from cardiac paralysis, cerebral hemorrhage, or arterial embolism before insufficiency occurs.

Aortic Stenosis.—Narrowing of the aortic orifice is rare, and is much less frequent than the other valvular lesions of the left side of the heart. It is very apt to be associated with aortic insufficiency. As a result of this lesion there is dilatation and hypertrophy of the left ventricle: the hypertrophy, however, being markedly predominant.

Aortic stenosis is represented by a loud, late, systolic murmur, heard with greatest intensity in the second right intercostal space and transmitted upward to the carotids. The murmur is also transmitted widely all over the chest. The aortic first sound is usually obliterated, the aortic second is much weakened, and, if there is also an insufficiency of the aortic valves, a diastolic murmur is heard.

The pulse is small, slow, and wavy, unless modified by insufficiency. Usually a thrill is heard and felt most distinctly in the aortic area and transmitted to the carotids. There are fewer general symptoms in this lesion of the heart than in any other, until the compensation, which is usually complete, gives way. When, however, compensation fails, the same general symptoms which have been described under aortic insufficiency occur. Eventually insufficiency of the mitral valve develops, with its concomitant signs and symptoms.

It is to be remembered that a systolic murmur in the aortic area is not distinctive of aortic stenosis, and also that an aortic systolic murmur may occasionally be heard with greatest intensity at the apex and be mistaken for that of mitral regurgitation. Other murmurs which may occur in the aortic area, and which are especially to be differentiated, are the functional murmurs of unusual distribution and the murmur of the very rare pulmonary stenosis. A patent ductus arteriosus may also produce a systolic murmur in the aortic area.

Tricuspid Insufficiency.—Endocarditis of the tricuspid valve acquired in extra-uterine life is very rare, and when it occurs is associated with lesions of the mitral or aortic valves or of both. Relative insufficiency of the tricuspid valve from stretching of the orifice occurs commonly as a result of mitral insufficiency and of such pulmonary diseases as emphysema and chronic bronchitis, which cause obstruction to the pulmonary circulation.

The chief signs of the regurgitation of the blood from the right ventricle into the right auricle are as follows. There is a wave of transmission which gives a systolic pulsation of the jugular veins, causing them to stand out prominently, especially on the right side. This systolic pulsation may also occur, although less frequently, in the liver from congestion of the branches of the hepatic vein. There is a systolic murmur, rather soft and low, heard at the lower part of the sternum, the maximum intensity being near the sternal junction of the fifth or sixth left costal cartilage, usually not distributed widely, but in some cases transmitted to the right as far as the axilla. The pulmonic second sound is, as a rule, not increased. The area of cardiac dulness corresponds to that of dilatation of the right ventricle, but is usually moderate, and extends to the right as well as to the left and downward. Cyanosis is especially pronounced.

It must be remembered that tricuspid insufficiency is in most cases exceedingly difficult to diagnosticate: in some cases there are no physical signs recognizable during life; in many cases the signs are those of the associated mitral lesion, and the pulmonic second sound remains strong even when a tricuspid lesion is found to be present.

Tricuspid Stenosis.—Tricuspid stenosis as an acquired disease in extra-uterine life is so very rare in children, so uniformly of congenital origin, and so very difficult of diagnosis, that it need only be referred to as a possible condition. Its diagnosis would depend on an enlarged right auricle, a presystolic murmur loudest near the ensiform cartilage, and venous congestion as in tricuspid insufficiency. Its frequent association with aortic and mitral stenosis makes its recognition, however, during life almost impossible.

Pulmonary Insufficiency and Stenosis.—Although organic disease of the pulmonary valve may result from fœtal endocarditis, it is rarely acquired in extra-uterine life, and then only in septic processes.

The diastolic murmur of pulmonary insufficiency should be distinguished from that of aortic insufficiency by not being transmitted to the carotids, by an hypertrophied right ventricle, by sharp accentuation of the pulmonic second sound, by evidence of septic embolism in the lungs, and by the absence of the Corrigan pulse.

When there is pulmonary stenosis the lesion is so universally of congenital origin that its diagnosis can be referred to what has already been described under congenital diseases of the heart.

Diagnosis.—The diagnosis of chronic valvular disease, when a single lesion of regurgitation or stenosis is present without an association with lesions of other valves, is made by the physical signs of the individual affection which have just been described. Combined valvular lesions are, however, exceedingly common, and render the diagnosis much more difficult. The diagnosis depends upon the character of the murmurs, upon the degree of dilatation and hypertrophy of the auricles and ventricles, and upon the general symptoms referable to the circulation and respiration. Too much importance must not be attached to the presence or sound of murmurs alone, as they may be functional. The distinction between organic and functional murmurs is given on page 748.

Prognosis.—Although children often show a wonderful power of recuperation, yet, unless the lesion is very slight, it can never be completely recovered from. The dangers from embolism or a recurrent endocarditis must always be borne in mind as increasing the gravity of the prognosis, but so long as compensation is efficient the prognosis is good, although murmurs of all kinds may be present.

Many of the influences which make the prognosis in later life bad, such as disease of the coronary arteries, are rare in early life. The mitral and aortic valves which are most commonly attacked in the acquired endocarditis of children are the ones in which compensation is most readily attained. In young children, however, the prognosis, on the whole, is unfavorable in chronic valvular lesions.

The nutrition of the rapidly growing heart is easily affected, and dilatation develops rapidly. Recurrent attacks of rheumatism are very common in children, and each attack increases the gravity of the prognosis. In like manner the onset of one of the acute infectious diseases means an exceptionally bad prognosis in children in whom valvular disease is present.

Extensive pericardial adhesions, insufficient or improper food, and lack of supervision of the amount of exercise greatly increase the risks of disturbing the compensation of the heart.

TREATMENT OF CHRONIC ENDOCARDITIS.—The indications for treatment of chronic endocarditis depend less upon the character of the lesion than upon the degree of compensation which is present. When compensation is established and withstands the ordinary demands of life, hygienic and prophylactic measures alone are necessary. As compensation fails, active treatment is called for, and is directly proportionate to the severity of the symptoms.

Simple hypertrophy of the heart, therefore, whether from endocardial causes, as from chronic valvular lesions, or from exocardial conditions, does not require the use of drugs. The regulation of the habits of life are of the first importance. Great care should be taken not to overtax any of the functions of the body. The digestive, nervous, and muscular systems should be kept well within the limits of fatigue. The diet should be carefully regulated according to the age and digestion of the child. Nervous strain from over-study and emotional excitement should be carefully guarded against. Out-of-door life and moderate exercise should be encouraged, but the more vigorous forms of athletic sports should be prohibited. Each case must be judged by itself as to the amount of muscular work which is permissible, and this can be determined only by beginning with the mildest forms of exercise and cautiously increasing the amount. It is often desirable to insist upon absolute rest in bed or on a lounge for an hour or two in the middle of each day.

With the advent of the early symptoms of failing compensation the cessation of all exercise and the use of appropriate doses of nux vomica and iron for a few weeks may restore the lost compensation. If the symptoms are persistent, the nux vomica should be supplanted by small doses of the tincture of digitalis. A change of climate is sometimes to be recommended in those cases in which the failure of compensation is slowly progressive.

Acute dilatation may come on suddenly with but few premonitory symptoms. The treatment is then absolute rest in bed and active stimulation by the use of strychnine and digitalis in doses as recommended on page 470. Brandy or aromatic spirits of ammonia are useful to bridge over a crisis, and not infrequently small doses of morphine, guarded with atropine, given subcutaneously, can advantageously precede the use of the cardiac stimulants. Digitalis and strychnine are, however, the drugs on which most reliance can be placed in all the stages of a broken compensation. As the cardiac weakness increases, special symptoms arise which demand special treatment.

Dyspnœa.—When the dyspnœa becomes troublesome, the child should be propped up by means of pillows or a bed-rest into a semi-upright position. The cause of the dyspnœa should be determined if possible, remembering that it may be due to flatulency, hydrothorax, hydropericardium, ascites, or œdema of the lungs. If it is of gastric origin, the digestive disturbance should receive appropriate treatment, both for the relief of the immediate symptoms and the prevention of its recurrence by the proper change in the diet. Hydrothorax, if present, may require repeated tapping, but this is of rare occurrence. The *œdema of the lung*, as evidenced by the dyspnœa, cough, and physical signs, is best controlled by stimulation of the heart rather than by treatment directed to the lungs. When the dyspnœa is apparently due to high blood-tension, the use of nitroglycerin, in doses such as are prescribed on page 470, will often meet the requirements.

Vomiting.—Vomiting is often a difficult symptom to control. If it is due to venous stasis, digitalis will often act well, but the vomiting may in itself be an early indication of the over-action of digitalis. If the vomiting is persistent, it may be necessary to omit all food by the stomach. Cracked ice in some simple charged water, or in champagne, or in milk and lime-water, can be used until stronger food is tolerated.

Dropsy.—The successful treatment of dropsy depends upon the recuperative power which remains in the heart. Digitalis is of the utmost use, and should be given in sufficiently large doses to produce a decided effect, but with careful attention to its physiological action. Its diuretic properties may be increased at times by giving it with small doses of calomel and caffeine, providing the kidneys show the evidence only of passive congestion. Saline cathartics can be given to diminish the œdema by depletion. It is often desirable to promote catharsis and diuresis on alternate days. If ascites or hydrothorax is excessive, the fluid should be aspirated at once, as delay may prove serious. The quantity of urine should be carefully noted when digitalis is being given. An increase in the quantity should follow its administration. When the urine diminishes steadily in spite of the digitalis, we should watch carefully for other symptoms of the cumulative action of the drug. Diuretin in doses of 0.3 to 0.6 gramme (5 to 10 grains) is a valuable diuretic in all forms of cardiac dropsy, and may be given alone or with digitalis.

Nervous Symptoms.—The nervous symptoms of restlessness and insomnia may, if moderate in degree, be controlled by trional or the bromides, 0.12 to 0.3 gramme (2 to 5 grains); more frequently they require the use of morphine combined with atropine, which are the most effective remedies we have, but require good judgment in their use.

Finally, it is of the greatest importance that a child with a chronic valvular lesion of the heart should be kept under medical supervision even during the intervals of complete compensation. By doing this we are frequently able to detect changes in the condition of the heart before the mother herself becomes aware of them, and by attention to the principles already outlined we may often avert the serious consequences which follow neglect of treatment.

FUNCTIONAL DISEASES OF THE HEART.

By functional disease of the heart we mean that there are no pathological conditions present beyond a weakness of the cardiac muscles and possibly, in some cases, a slight degree of dilatation of the cavities. It is a condition, therefore, represented by symptoms rather than by physical signs, with the exception that in many cases cardiac murmurs are present. It is rare to meet with functional cardiac disturbances before the middle period of childhood, but they increase in frequency as puberty is approached.

Etiology.—Functional cardiac disturbance may occur in the course of such neuroses as exophthalmic goitre. Again, it may arise from poor food and hygiene, from a lack of sufficient out-of-door exercise, and from overstrain in school life, leading to malnutrition with nervous exhaustion. It may also result from the use of such cardiac irritants as tea and coffee, and may follow or occur in the course of any of the acute diseases. The various forms of anæmia may produce functional murmurs, known as *hæmic murmurs.*

Symptoms.—The chief symptoms are palpitation, a weakened, irregular pulse, attacks of dyspnœa and fainting, and cardiac murmurs. These attacks occur in paroxysms and are not associated with the physical signs of organic cardiac disease.

Of especial interest are the cardiac murmurs of functional origin, as they simulate closely and must be differentiated from organic murmurs, whether congenital or acquired. These functional or, as they are sometimes called, hæmic murmurs are variable in character according to the position of the child, and may sometimes be intensified by the pressure of the stethoscope.

According to Jacobi, Steffen, and Hochsinger they may also be caused by the irritation resulting from deformity of the thorax such as occurs in rhachitis.

Diagnosis.—In the great majority of cases functional murmurs are systolic, soft, though they may be very loud, and are heard best in the pulmonic area. They are apt to be associated with anæmia, are evanescent, are not connected with cardiac enlargement, and are not accompanied by an accentuated pulmonic second sound or by general systemic venous engorgement.

While these functional murmurs may be transmitted in all directions, and are often associated with loud bruits in the vessels of the neck, yet, as a rule, they do not show their maximum intensity at the heart's apex and are not transmitted distinctly to the axilla and back. The systolic murmurs heard occasionally over the anterior fontanelle in infants are probably arterial.

In contradistinction to functional murmurs, organic murmurs are usually rougher and more musical, are distinctly transmitted to the great vessels of the neck or to the axilla and back, may be heard with maximum intensity over any part of the cardiac area corresponding to the various valves, are rarely loudest in the pulmonic area, and are usually associated with an enlarged cardiac area, accentuated pulmonic second sound, and in many cases with general signs of systemic stasis, such as cough, hæmoptysis, œdema of the lungs and extremities, ascites, and cyanosis.

PROGNOSIS.—The prognosis in the cases in which there is no organic disease of other organs, or when the disease in which they occur is benign, is exceedingly good, provided that the appropriate treatment is rigidly carried out.

TREATMENT.—During the attack mild stimulants, such as aromatic spirits of ammonia, should be given, and if there is a strong nervous element the bromides. In prolonged attacks digitalis in small doses is indicated, and in some cases strychnine.

When a cause can be detected, such as anæmia, it should be treated with iron or arsenic. When no especial cause can be found, all cardiac irritants should be avoided and careful directions given as to study, exercise, diet, and general hygiene.

The following case was one of primary malignant endocarditis with secondary infection of the pericardium, mediastinum, veins, lungs, pleuræ, and spleen.

A boy, four years old, was attacked one month previously with fever, thirst, and pain in his knees. Later his feet became painful and swollen, and other joints were successively involved. He complained of pain in the back of his neck and along his spine. One week before entering the hospital he began to have moderate but incessant choreic movements, and showed much incoördination of mastication and articulation.

A physical examination showed the lungs to be normal, the area of cardiac dulness somewhat increased to the left of the mammillary line, and a systolic murmur at the apex, transmitted into the axilla and back. On the following day a pericardial friction-sound was heard just above the left nipple, accompanied by precordial pain. Two weeks later the choreic symptoms disappeared, and the temperature became normal. The area of cardiac dulness did not extend under the sternum, but was found to correspond to the impulse of the heart, which was 1.4 cm. (½ inch) outside of the left mammillary line. During the last week of its life the child became very weak, had marked dyspnœa, and showed signs of effusion in the right pleural cavity, but presented no other symptoms.

The post-mortem examination showed that both pleural cavities contained considerable blood-stained fluid, in some places there were adhesions of the parietal and visceral layers, in others there was slight fibrinous exudation over the surfaces of the lungs. On section both lungs showed congestion and areas of broncho-pneumonia. The anterior mediastinum was injected, and some of the mediastinal glands were enlarged. The parietal layers of the pericardium and pleuræ were adherent and thickened. The heart was enlarged, and in places the myocardium was distinctly soft and pale. Along the free border of the right auriculo-ventricular valve there were a few fresh vegetations. The left side of the heart was dilated. The edge of the mitral valve was thickened and eroded. Small, whitish points could be seen beneath the endocardium, both on the walls and on the papillary muscles. The aortic valves showed a few fibrinous deposits at the edges of contact. The coronary arteries were normal.

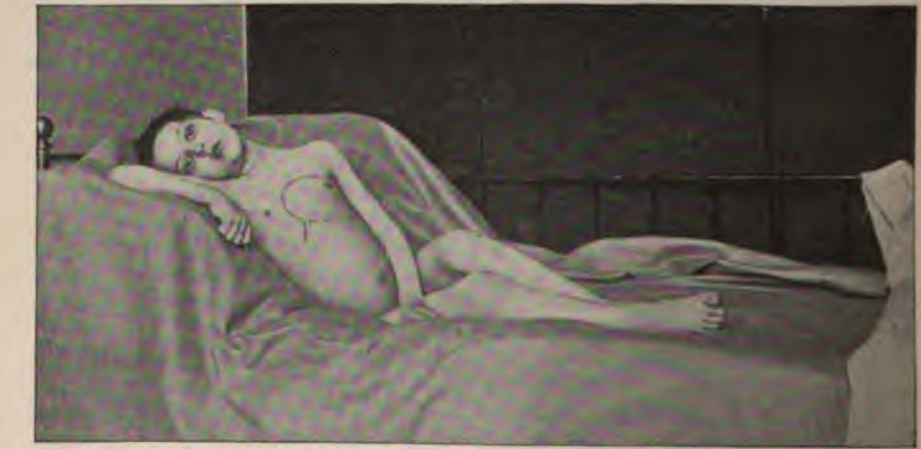
The spleen and lymph-glands were enlarged and soft. The liver showed congestion. In the left jugular vein was found an adherent thrombus which extended down into the subclavian vein, the innominate vein, and the superior vena cava, completely obliterating them.

Microscopic examinations showed the infection to be due to streptococci and the vegetations on the mitral valve to be distinctly verrucose. Pure cultures of streptococci were obtained from the lungs, pericardium, bronchial lymph-glands, and spleen; the other organs were sterile.

Dr. Councilman, who performed the autopsy, considered the endocarditis primary and of the malignant type, with a secondary infection of the lung. The mode of infection was probably from the heart to the pericardium, thence to the mediastinum, with thrombosis of the veins, from which the infection was carried to the lungs, setting up a broncho-pneumonia, which in its gross and microscopic appearances differed from the ordinary broncho-pneumonia of infants.

The next case illustrates simple acute endocarditis, followed by dilatation of the heart and loss of compensation.

Fig. 153.



Acute endocarditis. Mitral insufficiency. Lack of compensation. Orthopnœa. Female, 9 years old.

The child was nine years old, and although she had always been delicate she had never had any especial disease until two weeks previously, when she was attacked with fever, palpitation, cough, and a rapid, irregular pulse. On entering the hospital she was cyanotic, the face and extremities were cold, and there was considerable prominence over the cardiac region. The resonance of the lungs was normal, but there were a few moist râles at both bases. The impulse of the heart was in the fifth left interspace, 1.4 cm. (½ inch) outside of the mammillary line, and there was a marked thrill with a systolic murmur transmitted into the axilla and heard distinctly in the back. The liver was slightly enlarged. The temperature during the acute inflammatory stage of the endocarditis was moderately elevated, reaching 38.8° C. (102° F.) on the fourth day, and gradually subsiding.

The impulse of the heart was scarcely perceptible. The area of cardiac dulness extended to the right edge of the sternum, and slightly beyond the right sternal line beneath the third intercostal space.

The case illustrates an attack of acute endocarditis, either primary in character or more probably ingrafted upon a chronic endocarditis, which had never given rise to symptoms, as the limits of compensation had never before been disturbed. The acute inflammatory stage passed; dilatation took place, and there was marked failure of compensation, shown by the feeble impulse of the heart, the weak and fluttering pulse, the cold and blue extremities, the orthopnœa, and the tendency to œdema of the face, legs, and feet. Fig. 153 represents the position which the child assumed on her right side, supporting herself with her arms, and shows her anxious expression as she endeavored to keep herself in a position in which she could breathe easily.

The following case illustrates the condition of pure mitral stenosis, with periods of broken compensation alternating with periods of re-established compensation:

The boy, who was eleven years old, had measles when he was an infant, diphtheria when he was three years old, and pertussis when he was four years old. He had always been well until he was nine and a half years old, when, after indefinite pains in his joints, accompanied by no swelling and not sufficiently severe to confine him to bed, he began to have dyspnœa on exertion, and cardiac pain, cough, cyanosis, and considerable loss of weight. An examination of the heart showed the area of absolute dulness to be decidedly increased to the left. A loud presystolic murmur was heard at the apex, limited in its extent and accompanied by a thrill and a sharp first sound. There was no systolic murmur and no œdema. The physical examination showed nothing else of significance.

In the course of two months' treatment compensation was established, but the boy returned to the hospital from time to time with the same symptoms which have been described.

In the next case, in contrast to the one just given, we see the symptoms of a mitral stenosis combined with mitral insufficiency.

The child was a girl of thirteen years. When eleven years of age she had an attack of rheumatism followed by dyspnœa on exertion, and at times œdema of the feet. A week before I saw her she was taken with pain in the region of the heart, so severe that she could not sleep. On entrance to the hospital her temperature was 38.5° C. (101.2° F.), her pulse 104, and her respirations 65. The physical examination showed the apex-beat to be in the fifth interspace in the mammillary line. The area of cardiac dulness was enlarged, extending to the upper border of the third rib and about 5.3 cm. (2 inches) to the left of the mammillary line, and about a finger's breadth to the right of the right border of the sternum. A presystolic murmur was heard at the apex and was confined to a limited area, while a systolic murmur, also heard at the apex, was transmitted to the axilla and back. The pulmonic second sound was accentuated. In this respect the physical signs were negative, as, in spite of the marked hypertrophy and dilatation of the heart, compensation of the right heart was sufficient to prevent the results of venous stasis.

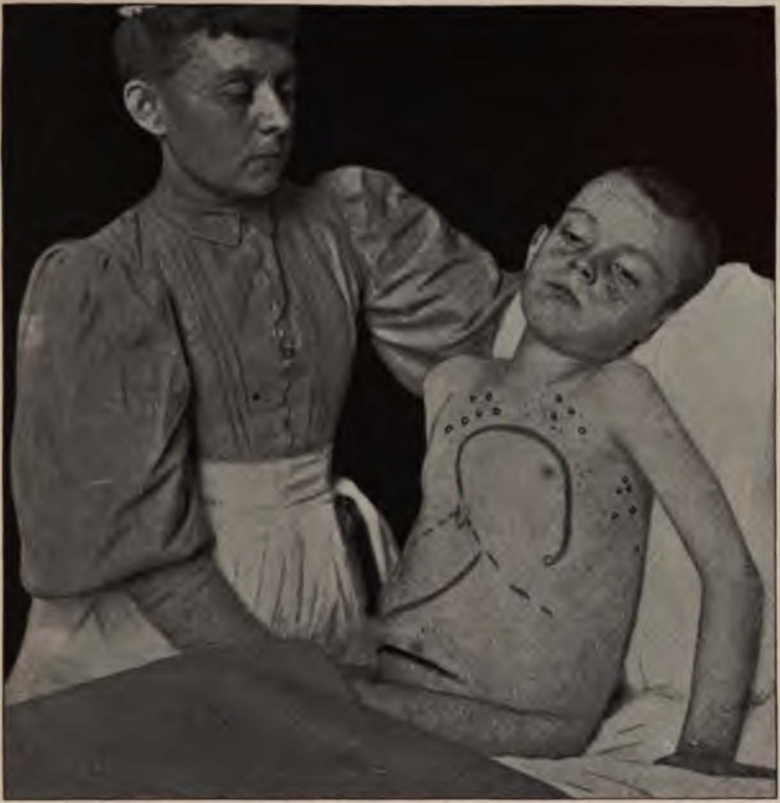
Four weeks later, after being treated by complete rest in bed, the cardiac symptoms almost entirely disappeared, the area of cardiac dulness was much diminished, the murmurs were less distinct, and the child left the hospital much improved.

The next case (Fig. 154), a boy, ten years old, is interesting as illustrating several characteristics of cardiac disease in early life.

When eight years old he entered the hospital with marked œdema of the face, body, and limbs, ascites, a slight amount of fluid in both pleural cavities, and œdema of the lungs. There was no definite history of rheumatism nor any other cause for the cardiac disease which was causing these symptoms, and which had apparently developed insidiously, although if he had been under closer observation a definite period of onset would probably have been discovered. The impulse of the heart was found to be 1.4 cm. (½ inch) outside of the mammillary line in the fifth left interspace. The area of cardiac dulness was somewhat increased. There was a loud systolic murmur at the cardiac apex transmitted to the axilla. The pulmonic second sound was much accentuated. Fig. 155 was taken at that time, and shows the marked œdema of the legs and the much distended abdomen. He was treated by complete rest in bed for five weeks, and in the beginning digitalis was administered until the urine, which was lessened in quantity, had increased and the œdema of the lungs had disappeared. On entering the hospital the ascites was removed by paracentesis abdominis. Under this treatment the child rapidly improved, the general œdema disappeared, the liver returned to its normal size, the area of cardiac dulness was markedly decreased, the cardiac murmur became less marked, and six weeks from the time when he entered the hospital complete compensation was established and he left the hospital seemingly perfectly well.

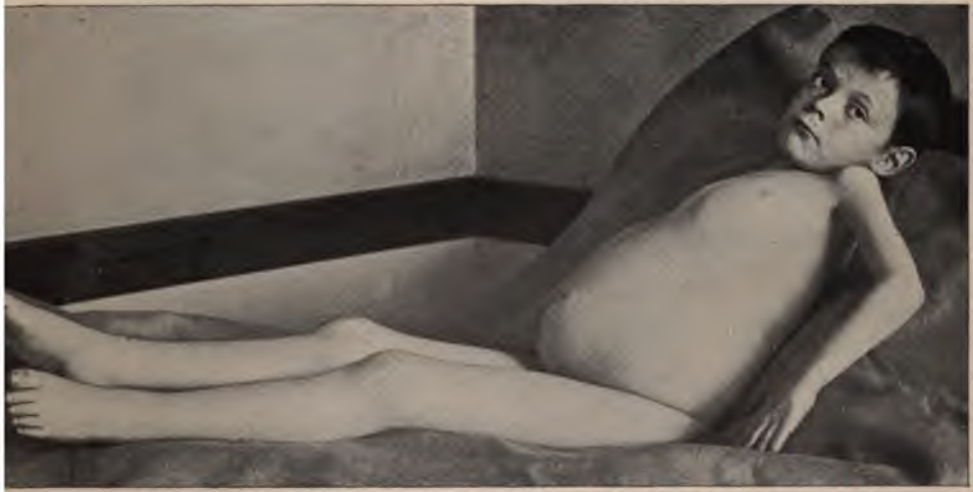
After leaving the hospital the boy was reported to have been very well, except that he could not play or work hard. Two weeks before his second entrance he was attacked with fever, precordial distress, and cardiac pain; later he began to have œdema of the feet and dyspnœa. From that time he grew progressively worse, and his case illustrates a fresh attack of endocarditis supervening on an old chronic endocarditis (endocarditis recurrens) and resulting in a disturbance of the previous compensation. He had orthopnœa to such an extent that he was unable to lie down in bed, and had to be continually watched by a nurse, as he frequently had attacks of excessive paroxysmal dyspnœa which were liable to prove fatal. There was cyanosis of the lips and hands and marked general œdema. The skin of the nose and extremities was cold. The impulse of the heart was felt in the sixth left interspace 2.8 cm. (1 inch) beyond the mammillary line. The area of cardiac dulness extended beneath the sternum, and at the third intercostal space extended 1.4 cm. (½ inch) to the right of the sternum, thence upward in a curved line across the upper part of the sternum to the second rib, and then, keeping outside of the mammillary line, descended and joined the point of cardiac impulse. There was a loud systolic murmur, heard most distinctly at the apex, but transmitted over the whole cardiac area and through the axilla to the back. The pulmonic second sound was accentuated. The aortic sounds were weak. There were numerous moist râles heard in all parts of the lungs. The percussion of the lungs was resonant everywhere except in the lower parts, where there seemed to be a slight amount of fluid in both pleural cavities. The liver was enlarged so that it extended 7.8 cm. (3 inches) below the margin of the ribs. Ascites was present, the fluid rising to about the line of the umbilicus. The spleen was normal in size. The child

Fig. 154.



Chronic recurrent endocarditis. Mitral insufficiency. Disturbance of compensation. Dilated heart. Enlarged liver. Œdema of lungs. Ascites. Male, 10 years old.

Fig. 155.

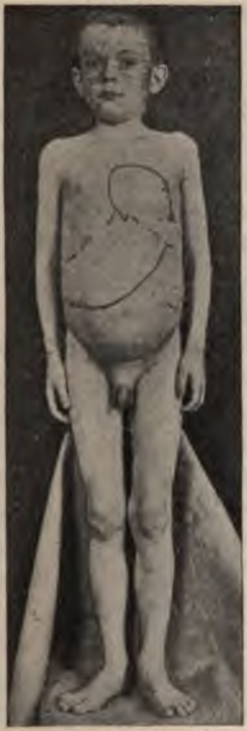


Chronic endocarditis. Mitral insufficiency. General œdema and ascites. (Before treatment.)

was passing only a small amount of urine, which contained a trace of albumin. The cardiac and hepatic areas of dulness and the upper border of the ascites have been marked by black lines, the margin of the ribs by broken lines, the point of cardiac impulse by a black ring, and the œdematous râles in the chest by smaller black rings. The prognosis in this case, although very serious, as he was liable to die suddenly at any time if extra blood-pressure should be brought to bear upon the dilated and crippled heart, was not entirely unfavorable, as he had previously shown such great powers of compensation and recuperation. As there was no great distention of the abdomen, the ascites was not removed by paracentesis. He was given infusion of digitalis, 3.75 c.c. (1 drachm), every three hours, and diuretin, 0.36 gramme (6 grains), once in six hours as a diuretic. His diet was milk.

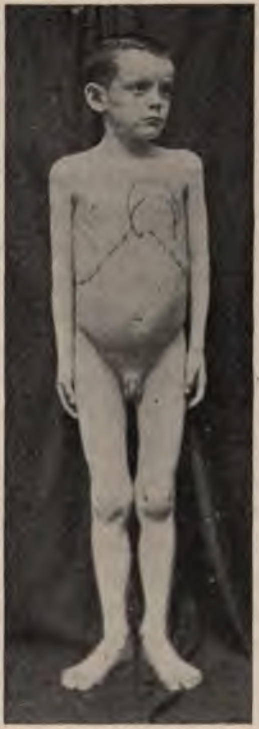
Within forty-eight hours rapid relief was obtained from the urgent symptoms, and at the end of three weeks the œdema of the lungs, the general œdema, and the ascites had disappeared entirely. The urine became normal in quantity and free from albumin. One week later he was well enough to be out of bed for an hour each day, but at that time the heart and liver were still enlarged, as shown in Fig. 156.

FIG. 156.



Chronic endocarditis. Mitral insufficiency. Returning compensation. Enlarged liver. Enlarged heart.

FIG. 157.



Chronic endocarditis. Mitral insufficiency. Broken line indicates enlarged heart. Black line indicates area of cardiac dulness with returned and complete compensation.

Some weeks later the liver regained its normal size, and still later the cardiac area of dulness was found to be much reduced and in the vertical line almost normal. Fig. 157 shows the enlarged heart, which persisted longer than the enlarged liver, and is represented by a broken line; the area of dulness of the heart as it appeared when he left the hospital is shown by a black curved line.

DISEASES OF THE PERICARDIUM.

The relations of the infant's pericardium, so far as I have been able to determine by the dissection of sixteen infants of different ages, does not differ from those of the adult. The amount of fluid which normally occurs in an infant's pericardium, although of variable quantity, is probably under 5 c.c. Hydropericardium, pneumopericardium, and hæmopericardium are so very rare that they need merely be referred to.

ACUTE PERICARDITIS.

Etiology.—Acute pericarditis may occur at any period, but the earlier the age the less often is it met with. It has been found in the fœtus and in the new-born. It is generally the result of an infective process, which may be primary or secondary. It may arise also as a result of direct extension of an inflammation from the pleuræ, mediastinum, and adjacent tissues.

Various bacteria have been demonstrated in the inflammatory products of pericarditis and in the pericardium itself, although they are not always connected in a specific manner with the disease. Different kinds of streptococci and staphylococci are found. The pneumococcus frequently occurs in cases following pneumonia, and is also found independently. Tubercle bacilli have been demonstrated in tubercular pericarditis, and the pericardium shows an especial tendency to the invasion of this bacillus following tuberculosis of the pleura. Tuberculosis as a primary disease is even more rare in the child than in the adult.

In the new-born, pericarditis may be the result of a septic condition following infection of the cord. At times it follows periostitis and ostitis in young children, here also probably being associated with septic infection. Traumatism may also be a cause of pericarditis. Rheumatism, especially after the third or fourth year of life, gives rise to as much peri-endocardial disease as at a later period. The inflammatory lesions may arise before the rheumatism has appeared elsewhere, and the intensity of the arthritic pain and the number of joints affected do not correspond to, or rather do not influence, the frequency of the pericardial complication. Inflammation of the pericardium is also frequently associated with pneumonia. It may be secondary to any of the eruptive fevers, but occurs most frequently as a complication of scarlet fever. When it occurs in this latter disease it appears usually in the second or third week of the attack.

Pathology.—Pericarditis may be circumscribed or diffuse, and there appears to be no essential difference between the pathological conditions affecting the pericardium in early life and those which occur later. The *pericarditis sicca* of the adult is comparatively unusual in the child, in whom, as a rule, an exudation of greater or less extent almost always takes place. The exudation may be sero-fibrinous, hemorrhagic, or purulent. Not only is the tendency to exudation in the child greater than in the adult, but its formation is characterized by greater rapidity and is more likely to be purulent. A pericardial exudation tinged with blood is not uncommon in early life, and is not necessarily so significant of tuberculosis as is a pronounced hemorrhagic exudation.

Acute Plastic or Dry Pericarditis.—SYMPTOMS.—The symptoms of acute plastic or dry pericarditis are often so mild as to be overlooked. There may be slight precordial pain and pyrexia. The characteristic physical signs are represented by a double to-and-fro murmur, which is not exactly synchronous with systole and diastole, is more superficial than an endocardial murmur, and is of a rubbing or grating character. The sound is localized in a small area, is not transmitted as in endocardial murmurs, and does not replace the heart-sounds. The murmur is at times intensified by pressure with the stethoscope. These signs, however, are of temporary duration, as a rule, as the exudation of fibrin is soon followed by that of fluid. In many cases in early life friction-sounds and endocardial murmurs simulate each other very closely.

Acute Pericarditis with Exudation.—SYMPTOMS.—The subjective symptoms of acute pericarditis with exudation in infancy are very indefinite, and throughout childhood this latency of the early symptoms is so marked and occurs so frequently that it may be said to be characteristic of the symptomatology of pericarditis in early life. It is so difficult to locate pain when it occurs in the infant, and a tumultuous action of the heart with general circulatory disturbance is so commonly the result of a diseased condition outside of this central organ, that it is impossible to formulate a practical general symptomatology for the onset of the disease. When, however, the disease has progressed, dyspnœa and orthopnœa become marked. Large exudations appear to affect the functional activity of the heart more rapidly in children than in adults, and to occasion earlier the signs of disturbance of the circulation, such as cyanosis and coldness of the nose and extremities. Diminution in the amount of the urine, with a corresponding increase in the urine as the exudation decreases, has been noticed in children.

PHYSICAL SIGNS.—The usual physical signs supposed to be characteristic of pericarditis are often very misleading, and when a pericardial friction-sound is absent the diagnosis of pericarditis in a young child may present great difficulties.

Inspection.—Owing to the flexible thorax of the child, there is a greater opportunity for the neighboring parts to yield before the pressure of the fluid, and we are more likely to have bulging of the intercostal spaces, and on inspection a visible alteration of the cardiac area, than in adults.

It has been held by certain authorities that the heart's apex is found in effusions to be tilted upward and inward towards the sternal end of the fourth left interspace,—that is, floated by the effusion. Direct proof of this is wanting, and I believe, from my investigations on this subject, that it is an erroneous view. It would seemingly be impossible for the heart to be floated unless the specific gravity of the fluid was greater than 1050, as I have shown by experiment. It is highly improbable that the specific gravity would be greater than 1050 in an ordinary pericardial exudation, for the specific gravity of a purely purulent fluid is only about 1032. Ludwig and Bowditch have, moreover, observed that the impulse of the heart as seen normally in the fifth left interspace need not be caused by the heart's apex, but may be caused by a portion of the heart above the apex striking against the thoracic wall. We should also consider that the impulse of the heart in children is often chiefly in the fourth interspace. It seems plausible to account for this pulsation by the tumultuous action of that portion of the right ventricle which, as shown by Rotch, is not covered by the moderate amount of fluid in the fourth left interspace when an exudation is present.

In the third case on page 761 it is recorded that the impulse was found throughout the whole cardiac area, but that it was still pronounced in the fifth left interspace. If in this case there had been a larger exudation, the apex and the lower segment of the right ventricle being surrounded by a mass of fluid, the impulse would have been lost in the fifth interspace, while in the fourth interspace, in which the ventricle may be covered by only a thin layer of overlying fluid, the impulse would have continued to be both seen and felt, thus simulating an apex-beat. I believe this is the explanation of what has been called misplaced apex-beats and floating of the heart in pericardial effusions.

Palpation.—When the amount of fluid is considerable the palpable impulse is much diminished, and its intensity corresponds to the place in which the visible impulse is most distinct.

Auscultation.—The friction-sound is often absent, but if present in the beginning disappears as the fluid increases, and may reappear when absorption takes place. The heart-sounds are diminished in intensity, but may be heard most distinctly in the fourth interspace at the point of the visible impulse. On account of the small size of the child's thorax, the heart and pericardium are much nearer to the anterior surface of the thoracic cavity than in adults. This occurs both normally and in diseased conditions, especially when there is flattening, and thus levelling, of the chest. Under these latter conditions the heart and pericardium are brought in such close contact with the thoracic wall that on palpation the heart's impulse can be felt, and on auscultation the heart-sounds can be heard in a much more advanced stage of a pericardial effusion than would be possible in the adult with a proportionately large amount of fluid.

According to Ewarts, a pericardial exudation may give rise to a patch of tubular breathing about two inches below and slightly to the left of the left scapula, but the same sign may occur in pleuritic exudation.

Percussion.—Percussion is the most important physical sign, when the initial friction-sound has escaped detection, both for determining whether an exudation is present and as a guide to the prognosis and treatment. In exudations of exactly the same amount the area of dulness may differ, owing to the difference in the elasticity of the lungs and to the presence or absence of adhesions. The greater the elasticity of the lungs and the fewer the adhesions the more regular will be the outline of superficial dulness. This superficial dulness is determined by the retraction of the borders of the lungs, which withdraw from the chest walls as the fluid gradually distends the pericardium. The deep dulness is due to the distended pericardium, and this to a greater or less degree compresses the lungs, which may be held in position by adhesions. The infant, being less likely to have had previous lesions of the lung and pericardium, gives us the best opportunity for studying the outlines of a pericardial effusion, and the area of superficial dulness is the most valuable physical sign of effusion in infants and in young children.

The experiments (Keating's Cyclopædia of Diseases of Children) on which I have based my conclusions regarding the area of dulness in pericardial effusions were made on sixteen infants, in none of whom did adhesions exist. In all of these presumably typical cases absolute dulness was found to the right of the sternum. An instance of how the area of dulness varies in cases complicated with adhesions was given by a case in which, although the pericardium was much distended with fluid, it failed to show dulness to the right of the sternum, and the autopsy revealed adhesions binding the lung tightly to the right edge of the sternum. The effusion was behind the lung, which permitted resonance to be obtained over an area where, in an uncomplicated case with the same amount of effusion, there would have been dulness.

In addition to the difficulties of making a differential diagnosis arising from interference with the contractility of the lungs, such complications as pneumonia of the right lung, especially its middle lobe, pleuritic effusion on the right side, an enlarged liver, and an enlarged heart must be considered. When this pneumonia, or pleurisy, or hepatic enlargement is present, an effusion into the pericardium cannot be diagnosticated by means of percussion, but the associated disease can usually be readily determined by its especial symptoms. The differential diagnosis, on the contrary, from an enlarged heart, especially a dilated heart in which the murmurs may be absent, can often be made only by means of percussion.

The superficial dulness of the heart is determined not by the shape of the heart itself, but by the marginal lines of the lungs, varying according to their expansion or retraction. Moreover, the pericardium itself, whether distended with fluid or not, does not by its own shape, as has been delineated so often in the plates illustrating pericardial effusions, aid us materially in determining the shape of the area of superficial dulness in a pericardial effusion. This area is marked by the retracting or rather displaced borders of the lungs.

When from 70 to 88 c.c. ($2\frac{1}{3}$ to $2\frac{2}{3}$ ounces) of fluid are introduced into the pericardium of an adult, there is a slight increase in the vertical as well as in the transverse area of dulness. The curved line which bounds the area of dulness starts at the sixth rib, to the right of the sternum, passes upward to the junction of the fourth cartilage with the sternum, impinges on the lower part of the third left interspace, and then descends just outside of the mammillary line to the sixth rib, to pass inward until it meets the dulness of the left lobe of the liver. This line forms an irregular semicircle, with a shorter radius to the right of the sternum and a longer one to the left.

A small section of the dull area, corresponding to the junction of the fourth and fifth ribs with the left side of the sternum, is formed by the heart itself, which is free from effusion at this point, while the rest of the dulness is produced by the effusion. The layer of fluid is very thin all over the upper portion of the effusion in the region of the fourth rib and fourth interspace, while the mass of the effusion is in the lower part of the sac on each side of the sternum in the fifth interspaces, the larger part of the mass being on the left side.

The same conclusions as to the area of dulness were obtained with a proportionately small amount of fluid in an infant about two weeks old; and of sixteen injections of fluid into the pericardial sac of infants of various ages the areas of dulness were identical in all, and in all the lungs were normal and there were no pulmonary or other adhesions.

When the amount of fluid in the pericardial sac is large, the transverse area of dulness produced by the much distended pericardium is increased so that it extends farther to the right of the sternum in the fourth and fifth interspaces, and then, rising to the third interspace, it occupies a small area on either side of the sternum under the third, second, and first ribs and the second and first interspaces, the upper lobes of the lungs having retracted from beneath the sternum. As the effusion increases the lungs retract still more, and the upper curved lines of the effusion on either side of the sternum present areas with still greater diameters.

According to Ewarts, a pericardial exudation may at times give rise to a patch of dulness in the back represented by a sharply defined square area bounded by the ninth or tenth rib above and the twelfth rib below, on the right by the spine, and on the left by a vertical line from the angle of the scapula. Over this area the respiratory sounds are absent and the voice-sounds feeble. I have observed these same signs in an unreported case in which the dulness disappeared as the pericardial effusion was absorbed. This dorsal patch of dulness is present normally in children up to the age of four years, due to the large size of the liver at this age.

Ewarts has also given as a point in the differential diagnosis between pericardial exudation and cardiac dilatation, that in a dilated heart the curve of the right border of dulness is directed downward and inward towards the ensiform cartilage, while in a pericardial exudation the curve is directed either downward or downward and outward. This distinction is more easily made in moderate than in very large exudations, as shown in Fig. 158, page 760.

Diagnosis.—From what has been said regarding the latency of the general symptoms of pericarditis in childhood and the difficulty of interpreting the local symptoms, it will be readily understood how important it is to recognize any especial symptoms which may characterize the disease. Instances have been reported in which a distended pericardium was mistaken by experienced diagnosticians for an exudation into the left pleura. The diagnosis as to the character of the effusion, whether serous or purulent, can only be made by aspiration. In infants the fluid is almost uniformly pus; this is also the case in the majority of older children, except in rheumatic infections. The condition which most closely simulates a pericardial exudation, both in its general symptoms and in its physical signs, is a dilated heart.

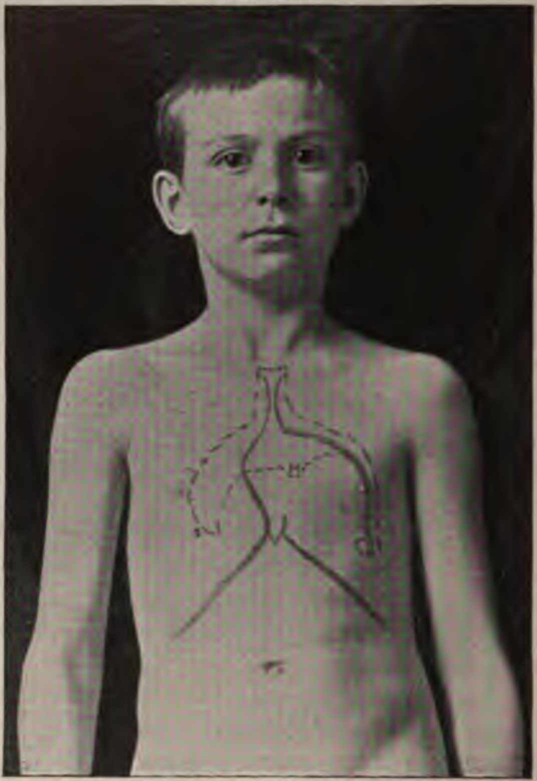
The most distinctive of all the physical signs of pericarditis is the friction-sound, when it is present, but it must be carefully differentiated from the sound produced by a dry pleurisy in the cardiac area. When, however, an exudation has taken place, the friction-sound may not be heard. This absence of a friction-sound is especially frequent in young children. The heart's impulse may be clearly perceptible, even when a considerable exudation is present, owing to the thin layer of fluid which covers the heart in the area between the left nipple and the sternum. We are therefore forced by the similarity which at times exists between the general symptoms, in the inspection, palpation, and auscultation of a dilated heart and of a pericardial exudation, to depend upon percussion in making a differential diagnosis.

According to careful observations which have been made by competent observers on the area of dulness which can be produced by an enlargement either of the ventricles or auricles of the heart, whether by hypertrophy or by dilatation, the relative dulness may extend to the right of the sternum from the second to the sixth rib, and in adults possibly to the distance of 3 or 4 cm. (1⅛ or 1½ inches) on a level with the fourth rib, but it is rare to find this dulness invading the fifth right interspace more than 2 or 3 cm. (¾ or 1⅛ inches). It is still more rare for the absolute dulness to be found in the fifth interspace at all, and even in the fourth interspace for more than 1.5 or 2 cm. (½ or ¾ inch).

We may conclude, therefore, that the dulness which occurs in a pericardial effusion may correspond to that of an enlarged heart, but that the dulness of the effusion is also found in an additional area corresponding to a part of the fifth rib and fifth interspace. Absolute dulness, therefore, in the fifth right interspace 3 or 4 cm. (1⅛ or 1½ inches) from the right sternal line in cases of pericarditis uncomplicated by pleural or pericardial adhesions is a sign of much value in the differential diagnosis from an enlarged heart. I have found in my experiments on the adult pericardium that the superficial dulness could be detected in the fifth right interspace when from 70 to 80 c.c. of fluid had entered the pericardium.

In order to illustrate the difference between the area of dulness produced by an enlarged heart and that produced by a pericardial effusion, I have indicated in Fig. 158, a boy, eleven and a half years old, the boundaries of the area of absolute dulness in an enlarged heart, in a small pericardial effusion, and in a large pericardial effusion.

FIG. 158.



Areas of absolute dulness in enlarged heart, and in distended pericardium. 5, fifth right interspace; H, heart.

The top of the sternum, the boundaries of the enlarged heart, the ensiform cartilage, and the lower border of the ribs are marked by plain black lines, the boundaries of the small effusion by a broken line, and the area of the large effusion by a larger broken line. The figure 5 marks the fifth right interspace; the letter H marks that portion of the heart which has been left uncovered by the small effusion. The small black circle represents the normal position of the apex of the heart, the larger circle the apex of the enlarged heart. It will be noticed how the enlarged heart extends beyond the right edge of the sternum at about the fourth rib and fourth interspace, and then returns beneath the lower part of the sternum within or a very little outside of the right sternal line.

The outline of the small effusion, as well as that of the large effusion, extends to the right of the sternum as low as the sixth rib.

The following cases, taken from a number which have come under my care, illustrate the difficulty of making a differential diagnosis between cardiac and pericardial disease when, as at times happens, we fail to find a friction-sound or murmurs:

TABLE 71.

Differential Diagnosis between a Dilated Heart and a Pericardial Effusion.

CASE I. Endocarditis; Dilated Heart.	CASE II. Pericarditis; Effusion.	CASE III. Endocarditis; Enlarged Heart; Pericardial Effusion.
Girl, eleven years.	Boy, six years.	Girl, eight years. August 3.
Attack followed acute articular rheumatism.	Attack followed acute articular rheumatism.	Attack followed acute articular rheumatism.
Orthopnœa; precordial pain.	Orthopnœa; precordial pain.	Orthopnœa; precordial pain.
Heart's impulse feeble, but perceptible a little to left and below left nipple, fifth interspace.	Heart's impulse feeble, but perceptible a little to left and below left nipple, fifth interspace.	Heart's impulse feeble, but perceptible all over cardiac area, with apex-beat a little below and to left of left nipple, fifth interspace.
Vertical absolute dulness not increased.	Vertical absolute dulness not increased.	Vertical absolute dulness not increased.
Absolute dulness under the sternum and to left of sternum; identical with Cases II. and III.	Absolute dulness under the sternum and to left of sternum; identical with Cases I. and III.	Absolute dulness under the sternum and to left of sternum; identical with Cases I. and II.
Absolute dulness did not extend to right of sternum.	Absolute dulness in fifth right interspace two or three centimetres from edge of sternum.	Absolute dulness in fifth right interspace three or four centimetres from edge of sternum.
Systolic murmur at apex.	Pericardial friction-rub at base.	Soft systolic murmur at apex, transmitted to axilla. Pericardial friction-rub at base.
Recovery.	Recovery.	August 6: Less dulness in fifth right interspace; apex murmur much louder and harsh.
		August 11: Dulness only to right edge of sternum.
		August 18: Dulness only to middle of sternum; friction-rub ceased.
		December 1: Physical examination the same as on August 18, showing enlarged heart and mitral systolic murmur.

The symptomatology, both general and local, of these cases was, with the exception of the friction-sounds, murmurs, and percussion, identical: when fluid was present dulness was found in the fifth right interspace, and when it was absent dulness was not found.

Prognosis.—The prognosis of acute pericarditis depends largely upon its cause. In early infancy it is a very serious condition, and generally ends fatally. In later childhood its course and results are much the same as in adults, and the disease has a tendency to recovery, especially if the exudation is sero-fibrinous and of rheumatic origin. The purulent, septic, and tubercular forms are of grave import in all cases.

Treatment.—The treatment of pericarditis in infancy and in early childhood does not differ materially from that in later life, and depends upon the various causes referred to in the etiology of the disease. The tendency to heart-failure, however, which is so pronounced in the child, should be guarded against. Early in the disease absolute physical and mental rest should be enforced. In the acute stage, before an effusion of any extent has formed, cold can be applied to the cardiac region by means of coils of tubing containing ice-water or by an ice-bag. An important part of the treatment is the judicious administration of digitalis to aid the heart in the crippled condition in which it is usually left after the early days of the disease. Stimulants should be freely used when there is any indication of failing compensation.

The most important part of the treatment when an effusion of any extent has occurred is paracentesis of the pericardium, which should unhesitatingly be performed, no matter what the cause of the disease may be, when life is in danger from undue distention of the pericardial sac. A small aspirating trochar should be used. Opinions differ widely as to the best point of puncture. Inasmuch as the heart, when an effusion is present, remains in its usual position, and does not, even when much enlarged, impinge on the fifth right interspace, and as the effusion, even when in so small an amount as 100 c.c., is found at that point, I consider it more rational to choose the fifth right interspace, 4 cm. (1½ inches) outside the right border of the sternum, as the point for tapping, thus avoiding all danger of injuring the heart. At this point the right internal mammary artery will not be injured. I have tapped the pericardium in the fifth right interspace a number of times on the cadaver, and have removed the fluid as easily as in the fifth left interspace. The pericardium has been tapped during life in the fifth right interspace by Ebstein, of Göttingen, Wilson, of Nashville, Lovejoy, of Lynn, and a number of others. Another place to aspirate, recommended by Osler, is the left fourth interspace, either close to the sternal margin or 2.5 cm. (one inch) from it, in order to avoid wounding the internal mammary artery. The left fifth interspace, 3.75 cm. (1½ inches) from the sternal border, may also be taken for the point of puncture, and if the aspirated fluid is found to be purulent, the case should be treated surgically by free incision.

As an illustration of how important it is to tap the pericardium when it is much distended with fluid and when symptoms of failing heart have arisen, the following case may be cited:

A boy, six years old, entered the hospital with the history of having had œdema of the face, hands, feet, and ankles for four weeks. There was no history of rheumatism, and the case was apparently one of acute primary endocarditis with mitral insufficiency. The cardiac area of dulness was increased, and extended from the middle of the sternum to 1.5 cm. (½ inch) beyond the left mammillary line, where the impulse of the heart could be felt. The child was kept quiet in bed, and after a few days the œdema lessened and he was very comfortable. While still under treatment, two weeks later, the temperature, which had been normal, rose to 39.1° C. (102.5° F.), the pulse was rapid and somewhat irregular, and the respirations were increased. A few days later a pericardial friction-sound was heard over the upper part of the sternum, and the temperature fell to 37.7° C. (100° F.). There was no change in the area of cardiac dulness, and no evidence of a pericardial effusion.

On the following day the cardiac sounds were found to be rather muffled and the child did not seem so well, and was unable to lie on his left side. Two days later the area of precordial dulness extended farther to the right, and a little beyond the right sternal line in the fifth right interspace. The attendants were directed to watch the child closely, and warning was given that the necessity for paracentesis of the pericardium might at any time arise. Early the following morning the child began to have marked dyspnœa and became very cyanotic. The house-officer found that the precordial dulness had extended 2.7 cm. (1 inch) beyond the right edge of the sternum in the fifth interspace, and he therefore got the instruments ready for performing paracentesis. Suddenly the child's pulse became very weak and intermittent, the cyanosis increased very much, the dyspnœa became very marked, and, although stimulants were quickly given, the child suddenly gasped and fell back on its pillow dead. This occurred within three-quarters of an hour from the time when the first serious symptoms arose. The house-officer immediately introduced the aspirating needle in the fifth right interspace and withdrew some fluid from the pericardium. The child, however, did not revive.

The following case illustrates a pericarditis sicca with adhesions:

The child was six and one-half years old. She had never had rheumatism, but had had an attack of measles when she was two years old, pertussis when she was two and one-half years old, and parotitis when she was three and one-half years old. Four months previous to her second entrance to the hospital she had an attack of chorea, of so mild a grade, however, that she was able to go to school until she entered the hospital. At that time, although she did not show any especial cardiac symptoms, an examination of the heart showed a latent and insidious endocarditis, represented by an increase of the superficial area of cardiac dulness to the left of the mammillary line, but not extending under the sternum, with a systolic murmur transmitted to the axilla, but not heard in the back. Compensation soon became complete, and she recovered from the chorea.

Two days before her second entrance to the hospital she was attacked with dyspnœa, rapid respirations, and cardiac pain.

On examination the child seemed very sick, and showed a considerable increase to the left in the area of cardiac dulness, and at the junction of the third rib with the sternum a marked precordial friction-sound, but with no enlargement to the right.

The precordial pain, discomfort, and heightened temperature lasted for a few days, and were in the beginning accompanied by orthopnœa and by an increase in the loudness of the friction-sound. There was at no time, however, any evidence of an effusion in the pericardium, and one week afterwards the friction-sound became less distinct, disappearing three days later. The child, however, grew much weaker, and, although she was treated by complete rest in bed and with digitalis, strophanthus, and stimulants, the precordial pain returned, and she gradually failed and died. The pulse varied from 130 to 150, and the respirations from 50 to 80. The temperature was only moderately elevated throughout the attack, ranging between 37.7° to 38.8° C. (100° to 102° F.) most of the time.

The autopsy showed the pericardial sac to be obliterated everywhere by firm fibrinous adhesions. The heart was enlarged. Along the edges of the mitral valve were numerous small grayish-white vegetations. These were also present on the aortic valves and on the portion surrounding the tricuspid valve. The lungs were denser than normal, and were deeply injected and œdematous. The pleura on the inner surface of the right lower lobe was adherent to the pericardium by fibrinous adhesions. The surface of the liver was covered with a thin layer of fibrin. The liver and kidneys were a little denser than normal, but were not noticeably congested.

The anatomical diagnosis was chronic adhesive pericarditis, acute vegetative endocarditis, acute fibrinous pleurisy, acute fibrinous perihepatitis, and hypertrophy and dilatation of the heart.

The next case was one of marked pericardial effusion, which was absorbed:

A little girl, eight years old, during the first two years of her life had scarlet fever, varicella, and pertussis. When she was two years old she had an attack of measles, and when she was seven years old an attack of chorea. During the last year she was fairly well until two weeks before entrance, when it was noticed that her feet began to swell, she complained of pain in her limbs, and occasionally of headache, she lost in weight, and had had orthopnœa with frequent paroxysms of dyspnœa. She also at times complained of pain in her left chest. Her extremities were apt to be cold. She had a short, dry cough. A physical examination showed the impulse of the heart to be feeble, but it could be felt all over the cardiac area. There was an area of precordial dulness extending to the right of the sternum almost to the right mammillary line, as low as and involving the fifth interspace and as high as the third interspace and to the left a little beyond the left mammillary line to the sixth rib. There was a systolic murmur at the apex, which was transmitted to the axilla. The pulmonic second sound was accentuated. There was a precordial friction-sound heard at the upper part of the sternum. The history of the case and the area of precordial dulness showed that it was a case of pericarditis with effusion. There may also have been some endocarditis, evidence of which is given by the mitral systolic murmur. The child was treated by rest in bed and by digitalis.

Two weeks later the friction-sound disappeared, and the precordial dulness grew less, so that it extended only to the middle of the sternum. In the next two weeks the dulness beneath the sternum disappeared, and the mitral murmur lessened, but could still be heard 2 cm. (¾ inch) outside of the mammillary line.

CHRONIC ADHESIVE PERICARDITIS.

Chronic adhesive pericarditis may occur in infancy and childhood. It is the result of acute inflammatory processes, in the course of which the parietal and visceral layers of the pericardium become adherent by the fibrinous exudation, which later becomes organized fibrous tissue. These adhesions may be partial or complete, involving the obliteration of the entire pericardial cavity. Well-marked adhesions of the pericardial surfaces have been observed in an infant who died thirty-six hours after birth.

If the adhesions are limited to the visceral and parietal layers of the pericardium, there may be no resulting cardiac disturbance, and in many cases there is neither hypertrophy nor dilatation.

If, however, an obliterated pericardium is associated with inflammatory adhesions between the outer layers of the pericardium and the pleura and chest walls, the hypertrophy of the heart may become extreme and eventually result in dilatation or sudden death.

The white, opaque thickenings of the inner pericardial surface so frequently found in adults are rare in children, but have been found at all ages, and when there is a deformity of the chest, as in certain cases of rhachitis, they have been especially noticed. The younger the subject the less likely are there to be adhesions between the pericardium and the pleura, an important fact, to be taken into consideration when making a diagnosis of pericardial effusion in infancy. The frequency of pericardial adhesions is shown in Lee's series of one hundred and fifty autopsies in cardiac disease of rheumatic origin, in which one hundred and thirteen cases had pericardial adhesions, and of these, seventy-seven had complete adhesion. An important result of pericarditis is the injury done to the myocardium through the intimate connection between the visceral layer of the pericardium and the heart-muscle. Lee in thirty-four of his cases found changes in the myocardium after pericarditis.

In the fibrinous pericarditis of rheumatic origin the process is likely to be of a very mild grade, with but a slight increase in the connective tissue between the membranes, and consequently with but little thickening. The most extreme cases of thickening of the pericardial membrane is met with in the chronic tubercular form. In these cases there is always a primary tuberculosis of the mediastinal or bronchial lymph-nodes. The tubercular process invades the pericardium by extension from these foci. This is not an uncommon process in children. Of sixty-five cases collected by Brackman, nineteen occurred in children.

After the absorption of an extensive purulent or sero-purulent exudation, the resulting thickening of the pericardium may be increased by the deposition of lime-salts within the membranes leading to calcification. Such areas of calcification are most frequently found over the auricles or at the base of the heart. According to Osler, the heart may be completely invested by a calcareous membrane which in places may be from 1 to 1.5 centimetres ($\frac{6}{16}$ to $\frac{9}{16}$ inch) in thickness.

SYMPTOMS.—The symptoms of adhesive pericarditis are not distinctive. They are generally dependent upon the condition of hypertrophy and dilatation which is present and upon the degree of compensation.

At times the symptoms are suggestive of cirrhosis of the liver,—that is, enlargement of the liver and ascites. When these occur without obvious cause in a young person, adhesive pericarditis should be suspected.

When severe cardiac symptoms are present in young children and no valvular murmurs are heard, we should think of degeneration of the heart-muscle itself or of pericardial adhesions. When, again, the superficial area of dulness remains unchanged and there are well-marked systolic retractions, the presence of pericardial adhesions is highly probable.

Diagnosis.—The chief diagnostic points of adherent pericardium associated with chronic mediastinitis, are bulging of the chest, systolic retraction at the cardiac apex, or in Traube's space, and systolic retraction in the line of the attachment of the diaphragm (Broadbent's sign). Associated with this retraction may often be seen a diastolic collapse of the cervical veins, the so-called Friedrich's sign. Systolic retraction, however, may occur in pronounced hypertrophy without adhesions.

An extreme dilatation of the heart with an adherent pericardium will give rise to signs which closely resemble pericarditis with exudation. The distinction between these two conditions can only be made by the very closest observation of the physical signs of pericardial exudation as described on page 755. The majority of the cases of adhesive pericarditis are not recognized during life, and are only discovered at the autopsy.

Treatment.—The treatment is symptomatic, the endeavor being made to maintain the compensation of the heart. When ascites is present, withdrawal by paracentesis may aid in the palliative treatment.

DIVISION XII.

DISEASES OF THE ŒSOPHAGUS, STOMACH, AND INTESTINE.

INTRODUCTION.

BEFORE speaking in detail of the diseases of the stomach and intestine, a few general remarks are necessary to explain how limited is our knowledge of these diseases. Those diseases, however, which affect the œsophagus can easily be classified on a pathological basis, and are so few in number that they can be included in these general remarks.

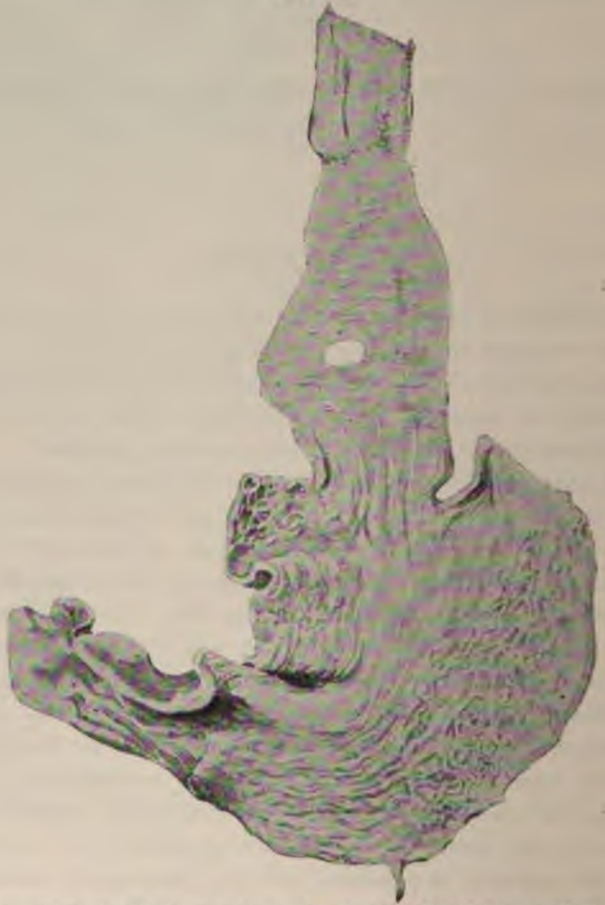
ŒSOPHAGUS.—The diseases of the œsophagus are rare in infancy and early childhood. There may be congenital malformations, such as absence or obliteration, narrowing or dilatation, and fistulous communication with various parts, such as the trachea, or that congenital condition called "branchial fistula," described on page 296. The swallowing of hot or corrosive liquids may cause obstruction, which is occasioned by a cicatricial stricture. Œsophageal stricture may also occur as a result of congenital syphilis. Pressure outside of the œsophagus may cause obstruction. These strictures, especially those of cicatricial origin, are accompanied by a great deal of muscular spasm, which at times is constant, and again relaxes. Thus, the child will swallow with comparative freedom at intervals, while at other times the obstruction appears to be complete. In addition to the inability to swallow, and the consequent regurgitation of the food, the secretion of saliva and mucus is often very profuse, and causes symptoms of distress and choking.

The diagnosis and treatment of these cases are effected chiefly by means of bougies; but, as much harm may come from these instruments, and as especial surgical knowledge is required to use them and to decide whether œsophagotomy should be performed, I shall not dwell on this class of cases.

An inflammatory condition of the œsophagus is said to occur in young infants, and is spoken of as *œsophagitis*. It is rare. The symptoms, as described by Billard, are unwillingness to nurse, crying, immediate regurgitation after beginning to suck, and often some tenderness about the neck on pressure. The prognosis is bad. The treatment of the active stage of the more severe forms, such as the corrosive, is by the administration of emollients and ice. Morphia may be required for the pain.

It is quite common for children to swallow various *foreign bodies*, such as buttons and pins. These bodies may either be caught in the back of the throat or lodged in the œsophagus, instead of passing through to the stomach. A careful examination of the throat with the finger should first be made, and if the foreign body is not detected in the throat the œsophagus should be explored carefully with a bougie, and the foreign body is then usually pushed through into the stomach, unless it is thought wiser to remove it with the bristle probang. The diet for the following twenty-four to forty-eight hours, or until the body has been passed through the intestine, should be such as will give sufficient consistency to the fæces to protect the intestine from injury while the body is being passed over its surface. Various preparations of the cereals are useful for this pur-

Fig. 159.



Congenital dilatation of œsophagus, female, 10 weeks old (⅗ natural size).

pose. If necessary, a dose of oil can be given, but, as a rule, active treatment is contra-indicated.

The œsophagus and stomach of an infant ten weeks old, with congenital dilatation of the œsophagus is shown in Fig. 159.

The infant was healthy at birth, and its mother had a plentiful supply of breast-milk. During the first two or three weeks of its life nothing abnormal was noticed about it, except that it vomited occasionally. When it was four weeks old it began to regurgitate, vomited the milk frequently, and lost in weight. The fæcal discharges showed that the milk which reached the stomach and intestine was fairly digested, but the discharges were infrequent and small in number. It was weaned when it was nine weeks old, and small amounts of milk, carefully modified in various ways, were given to it. No improvement in the symptoms followed this treatment, and although at times a small quantity of milk would be retained, yet, as a rule, after a few minutes the milk was regurgitated. The infant had no other symptoms, but rapidly lost in weight, and finally died of exhaustion.

The post-mortem examination was made by Dr. Whitney, and the only pathological conditions found were in the œsophagus. . The last two inches of the œsophagus were dilated into a more or less cylindrical swelling, with marked thinning of the walls and atrophy of the mucous coat. A dilatation had been formed in which evidence of a small area about to perforate into the mediastinum was found. The entire stomach, as well as its cardiac and pyloric orifices, was markedly contracted, apparently from lack of use.

STOMACH AND INTESTINE.—Our knowledge of the diseases of the stomach and intestine is exceedingly limited, and is especially so when infants and young children are concerned. The classification of these diseases on a pathological basis has been proved to be inadequate, and in like manner a classification on the basis of symptoms is insufficient. Bacteriological investigations, however, have advanced our knowledge to such an extent that we may hope in the future to be able to classify these diseases on an etiological basis. The terms dyspepsia, dysentery, diphtheritic, croupous, and others have become almost unmeaning, and should be replaced by terms more closely connected with the etiology of the disease.

In 1894 the American Pediatric Society requested Dr. Holt and myself to prepare a nomenclature which would correspond more nearly to our present knowledge of this exceedingly difficult subject. I wish especially to emphasize the value of Holt's work, which has aided me so much in my own studies on this subject. The classification finally adopted by the Society was one which especially relates to infants and young children, and it is this early period of life that is about to be described. The diseases of the gastro-enteric tract as they occur in older children resemble so closely those of adults that they do not occupy a prominent place in the clinical medicine of children, especially as the pathology and symptoms of this later period of life differ very materially from those of the earlier periods. These differences are still more strongly marked from the fact that children succumb much more readily to the early stages of a disease than do adults, and may die before the later and more characteristic lesions and symptoms of the disease have developed. There are certain known facts resulting from the anatomical and physiological peculiarities existing in infancy which play a significant part in all these diseases. It is well, therefore, first to explain the general principles which influence the symptoms and prognosis of these diseases before attempting to describe each disease separately. In many cases we can arrive at only approximate conclusions as to the actual lesion which exists and the prognosis which should be given. A practical clinical diagnosis should be made according to the region in which the stress of the lesion exists, rather than to all the pathological lesions which are present.

GENERAL ETIOLOGY.—In the present state of our knowledge it is not practicable to discuss in detail the various supposed causes of gastro-enteric disturbances. We can suppose that these disturbances may be due to nervous conditions which may act alone or may render the tissue vulnerable to bacteria. Some of these diseases are caused by specific organisms, while others are due to a number of organisms. These bacteria act either of themselves or through their products.

In a general way, these diseases can be classified as functional and organic. The organic class may be divided into inflammatory and non-inflammatory diseases, although the boundary-line between these two conditions is at times very doubtful. A prominent and important peculiarity of these diseases as they occur in infancy is, as would naturally be expected at this early period of development, a variety of symptoms which are produced by reflex causes. By the term reflex we mean peripheral irritation with a resulting action. By functional we mean a disturbance of the function of the organ without a known lesion. By organic we mean a known lesion.

In addition to these cases are others which, as yet imperfectly understood, seem to be produced by certain morbid products eliminated from the blood by the gastro-enteric tract, as, for example, urea. This etiological factor can be spoken of under the term *eliminative*.

GENERAL PATHOLOGY.—The general pathological anatomy of the gastro-enteric tract of infancy and early childhood is essentially that of the ileum and colon. In those cases in which the more severe lesions are present the stress of the lesion is usually in the lower ileum and the colon, and very frequently in the colon only. For this reason the terms ileo-colitis and colitis seem more descriptive than ileo-enteritis and enteritis. The pseudo-membrane in ileo-colitis is often extensive, but sloughing and perforation are exceedingly rare in young children. It is at present believed that not all ulcers of the gastro-enteric tract are necessarily inflammatory. The great number of lymph-nodules and the abundance of the lymphatic plexuses are the principal anatomical conditions which influence the pathology of the enteric tract in early life.

GENERAL BACTERIOLOGY.—The knowledge of the different bacteria which occur in the gastro-enteric tract, and of the connection which they have with the different diseases, is at present, with few exceptions, uncertain and unreliable. The proteus group has, however, been most commonly found in cholera infantum, while in the cases of fermental diarrhœa the ordinary saprophytic bacteria become prominent, especially the hay bacillus. These bacteria usually enter the system through milk, but may enter through other sources. There is little doubt that the bacteria may find their way, by means of the stomach, to the intestine, and that the acid secretion of the stomach which they meet in their way through it is not sufficient to prevent their arriving alive in the intestine. We know that these bacteria play such an important rôle in their etiological relations to the various diseases that full weight must be given to their presence when we are treating the disease. It would seem that the bacteria which are commonly found in the intestine when it is in a normal condition do not cause any abnormal conditions; but when the intestine has become irritated, from mechanical or thermic causes, the bacteria are able to penetrate its mucous membrane, become noxious, and produce abnormal symptoms, often of a serious nature.

General Symptomatology.—Vomiting as a symptom is often very misleading in early life, so far as the differential diagnosis between the stomach and the intestine is concerned, as it frequently occurs from disturbance in any part of the gastro-enteric tract, and should not be considered as indicative of any one disease. Serious symptoms during life are often proved at the autopsy to have been produced by no pathological lesion, while grave lesions may be found at the autopsy where the intestinal symptoms during life were very mild.

Marked diarrhœa may exist during life and no lesions be present at the autopsy. Serious lesions may exist, and yet no blood appear in the dejections. Blood may appear in the dejections, and yet no serious lesion exist, the hemorrhage being only temporary, and comparable to epistaxis.

General Diagnosis.—The observation of the temperature is very important for the diagnosis of these diseases. As a rule, an elevated temperature of short duration points towards functional and toxic disturbances, while an elevated temperature long continued points towards inflammatory lesions. The chemical examination of the gastric contents in infants has so far proved to be unsatisfactory both for diagnosis and treatment.

Intestinal discharges are often very misleading in making a diagnosis.

Having considered and accepted these general principles relating to diseases of the gastro-enteric tract in infancy, the American Pediatric Society adopted the classification, as presented to them by their committee. This classification must be understood to be merely provisional, and is for the purpose of aiding those who are interested in this subject to work with uniformity.

At the same time it is believed that it is a great advance upon the unmeaning and misleading nomenclature now current. Since the meeting of the Pediatric Society in 1894 certain diseases, such as cholera infantum and tubercular ileo-colitis, have been transferred from the gastro-enteric to infectious diseases as manifestly being general rather than local.

On examining the classification (Table 72, page 773) it is seen that whenever the etiology has been definitely determined it is made to designate the disease, but the true etiology is still unknown in so many cases that other terms have of necessity been used, the names simply representing the extent of the knowledge we have of the especial disease.

The diseases of the gastro-enteric tract may, on this basis, be divided into diseases of the stomach (gastric), diseases of the intestine (enteric), and the disturbances which arise from animal parasites. The diseases are then divided into those which arise from developmental, those which arise from functional, and those which arise from organic causes. The organic diseases are subdivided into non-inflammatory and inflammatory, and the functional and organic diseases into acute and chronic.

GENERAL TREATMENT.—In the treatment of these diseases we should endeavor to carry out four rules: (1) to combat the nervous symptoms if causative; (2) to dislodge the bacteria as quickly as possible, perhaps by laxatives and irrigation; (3) not to introduce into the gastro-enteric tract for a certain period food which may prove a favorable culture-ground for the bacteria, since it has been shown that where the food is sterile when it enters the gastro-enteric tract it is quite effective in reducing the number of bacteria in the intestine; (4) to introduce such drugs into the gastro-enteric tract as may, by their anti-fermentative and germicidal powers, diminish the action of or destroy the bacteria. This last rule is, however, very difficult to carry out, and, with our present knowledge of drugs and their administration, practically impossible. It is true that we know that subnitrate of bismuth is an anti-ferment, and that it reaches the part of the enteric tract which we know to be most affected in enteric disturbances characterized by fermentation. Fig. 160 shows the intestine of an infant, given to me by Dr. Holt, to illustrate this point, to whom bismuth had been given, and in whom at the autopsy the bismuth was found thickly coating the mucous membrane of the small intestine, and also appearing in the large intestine. It is, however, questionable whether in any case the attempt to kill the bacteria by the internal administration of drugs has been successful. Preparations, such as salol, which are known to be broken up into their carbolic acid components on reaching the intestine, cannot with safety be given to the infant in doses large enough to kill the bacteria, for in such doses there may be serious results from poisoning. We can, however, possibly, by means of these germicidal drugs, produce a condition in the intestine which, though not conducive to the death of the bacteria, may yet be so unfavorable for their growth as to aid our treatment when we are endeavoring to dislodge them. Nothing definite has, however, as yet resulted from using drugs for this purpose, and, so far as I can judge, the danger of treating infants or young children in this way is greater than the good that may result from it.

TABLE 72.

Diseases of the Gastro-Enteric Tract.

- A. GASTRIC.
  - I. DEVELOPMENTAL.
    - (*a*) Malformations.
    - (*b*) Malpositions.
  - II. FUNCTIONAL.
    - (*a*) Acute.
      - (1) Nervous vomiting
        - Central-Reflex.
      - (2) Cyclic vomiting.
      - (3) Indigestion.
    - (*b*) Chronic
      - Indigestion.
    - (*c*) Eliminative.
  - III. ORGANIC.
    - (*a*) Non-Inflammatory.
      - (1) Mechanical.
      - (2) Ulcers.
      - (3) New Growths.
    - (*b*) Inflammatory.
      - (1) Acute Gastritis
        - (*a*) Catarrhal.
        - (*b*) Corrosive.
        - (*c*) Pseudo-membranous.
      - (2) Chronic Gastritis
        - Catarrhal.
- B. ENTERIC.
  - I. DEVELOPMENTAL.
    - (*a*) Malformations.
    - (*b*) Malpositions.
  - II. FUNCTIONAL.
    - (*a*) Acute.
      - (1) Nervous.
        - Central-Reflex.
      - (2) Indigestion
        - (*a*) Duodenal.
        - (*b*) Intestinal.
    - (*b*) Chronic.
      - (1) Nervous.
        - Central-Reflex.
      - (2) Indigestion
        - (*a*) Duodenal.
        - (*b*) Intestinal.
      - (3) Incontinence.
      - (4) Constipation
        - Atonic-Spasmodic.
    - (*c*) Eliminative.
  - III. ORGANIC.
    - (*a*) Non-Inflammatory.
      - (1) Fermental
        - Acid-Albuminous.
      - (2) Mechanical
        - Dilatation of Colon.
        - Volvulus.
        - Intussusception.
        - Hernia.
        - Fissure.
        - Prolapse.
        - Polypi.
        - Hemorrhoids.
        - Fistulæ.
      - (3) New Growths.
    - (*b*) Inflammatory.
      - (*a*) Proctitis.
      - (*b*) Appendicitis
        - (*a*) Acute.
        - (*b*) Chronic.
      - (*c*) Ileo-Colitis
        - (*a*) Acute.
        - (*b*) Chronic.
- C. ANIMAL PARASITES.

DISEASES OF THE STOMACH.

GENERAL CONSIDERATIONS.—From what has been said it will be understood how difficult it is to make a differential diagnosis between gastric and gastro-enteric disturbance. The only symptom which definitely shows the stomach to be involved, whether from reflex, functional, or organic conditions, is vomiting, and, as we know that in many cases vomiting is caused primarily by disturbance of the intestine, we really have no symptom which represents gastric disease alone. The difficulty of locating disease in the stomach is rendered still greater by the fact that serious organic lesions may exist in the stomach without any symptoms whatever, whether of vomiting, pain, or tenderness. We must, therefore, be exceedingly cautious in making a diagnosis of diseases of the stomach.

Diseases of the stomach may arise from *developmental*, *functional*, or *organic* causes.

I. DEVELOPMENTAL.—Under developmental affections of the stomach are included *malformations* and *malpositions*. A malformation of the stomach may be represented by a narrowing of either the cardiac or the pyloric orifice, or by constrictions in various parts of the ventral cavity, which are known as hour-glass contractions. A malposition of the stomach may be met with in various places, one of which is in the thoracic cavity. These malpositions, however, are exceedingly rare, and of pathological rather than of clinical interest, as the diagnosis can scarcely be made during life. Congenital stenosis of the pylorus in infants, resulting fatally in a few weeks or months, has been described, but its existence has been questioned.

II. FUNCTIONAL.—The functional diseases of the stomach play a great rôle in infants and in young children. They may be of an *acute* or a *chronic* variety, or may be what I have spoken of as *eliminative*. Acute functional gastric symptoms may be produced by a number of causes, but in general they are to be understood as arising from a *nervous* condition represented by vomiting or by a disturbance of the function of digestion, which had best be spoken of, until more is known of the subject, as *simple indigestion*.

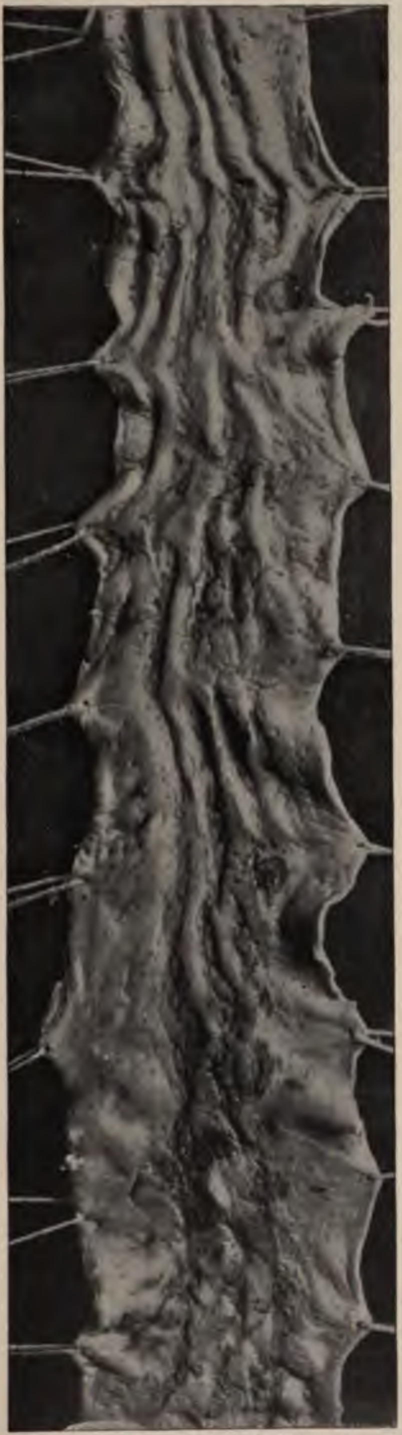
ACUTE NERVOUS VOMITING.

Vomiting may arise from gastric or from intestinal irritation in many diseases, such as tubercular meningitis, from heat, cold, fright, and from other causes. Direct irritation, from foreign bodies, food, or otherwise, may produce a reflex form of vomiting. In these cases the cause, if possible, should be removed, and the stomach given a complete rest until the nervous disturbance has subsided. As a rule, no internal remedies are indicated in these cases, except an emetic when the vomiting arises from the reflex causes just described, or, if necessary, lavage.

CYCLIC OR PERSISTENT VOMITING.

There is one form of vomiting which is of such importance that it must be spoken of as a disease by itself. It has been called cyclic or persistent vomiting, as no single definite cause nor any pathological lesion has ever been proved to produce it. It has not even been shown that it is a primary disturbance of the stomach. In fact, in many cases it is possible that the source of irritation is entirely outside of the stomach, and perhaps connected with the great sympathetic ganglia, such as the solar plexus, or

Fig. 160.



Colon showing presence of bismuth which had been given by the mouth.
(Page 772.)

it may eventually be proved to be of toxic origin and be placed under the eliminative class.

Etiology.—The inciting cause of the vomiting in most cases is obscure, but is evidently very varied. It does not seem to be produced especially by errors of diet, but, on the contrary, occurs in children whose diet has been most carefully regulated. Undue exposure to cold, fright, and excitement all appear to me to have sometimes an etiological influence on the disease. This form of vomiting may occur at any age. I have met with cases in young infants and throughout the whole period of childhood. The attacks may occur not only in delicate, nervous children, but also in those who are quite vigorous.

Symptoms.—The attack is very apt to come on suddenly, the child being previously in good health and not having shown any digestive disturbance. The period over which the disease extends and the intervals of the vomiting during the attack vary considerably, but in those cases which have come under my notice they are somewhat as follows. The child, without any especial warning, begins to vomit, and at first the vomitus will simply be the remains of food which still happen to be in the stomach. It will continue to vomit quite regularly every fifteen or thirty minutes. This may last for ten or twelve hours; the intervals then grow longer, and sometimes the vomiting will cease for twelve or fifteen hours, and then begin again. Occasionally a little mucus appears in the vomitus, but not to any great extent. As the disease progresses, a slight amount of bile usually appears in the vomitus. A very prominent symptom is thirst, the child crying continually for water, and vomiting it soon after it is taken. As a rule, the temperature in these cases is normal or subnormal. The pulse varies, but is very apt to be slow, sometimes intermittent, and may become weak. After the first twenty-four hours the child emaciates rapidly, looks very ill, and becomes apathetic.

Unless the disease is unwisely treated by endeavoring to introduce food or drugs into the stomach, it will usually prove to be self-limited, and will run its course in two or three days. In some cases the length of the attack is much shorter, being comprised within twenty-four hours, while in others it may last for many days. The recovery is often as sudden as was the onset of the disease. As soon as the vomiting has stopped, the appetite returns; there are no special symptoms of indigestion; the child takes its food well, and the emaciation disappears rapidly. Relapses occasionally take place.

Diagnosis.—The diagnosis of this form of vomiting is often difficult, more on account of a lack of sufficient knowledge concerning the disease than from much evidence of the existence of the diseases which it is supposed to simulate. Thus in early infancy acute persistent vomiting may be the first symptom of a beginning tubercular meningitis. In these cases an examination of the abdomen should be made at once, including a rectal examination. This is necessary in order to exclude such sources of vomiting as intussusception and appendicitis. The absence of any marked increase in the temperature and a careful examination of the thorax will in most cases exclude the sudden onset of some pulmonary disease or of the acute infectious diseases. The disease which is most commonly suspected in these cases is tubercular meningitis. In some instances, after the disease has lasted for two or three days, the resemblance to tubercular meningitis may be quite striking; but if the whole course of the affection is taken into consideration, the diagnosis soon becomes clear. In cyclic vomiting the face and general appearance of the child indicate nausea rather than the apathy which would be present in tubercular meningitis. The mind also, in contradistinction to what takes place in the latter disease, is clear, the child remaining quiet merely because it is exhausted. The great thirst which has been already mentioned as being present in persistent vomiting also aids materially in the differential diagnosis from tubercular meningitis. The sudden onset of the vomiting in a previously healthy child in the middle period of childhood is quite different from the slow progress and the occasional vomiting of a cerebral type met with in tubercular meningitis.

After the first twenty-four hours, this disease is readily differentiated from attacks of simple indigestion, for, when the vomiting arises from indigestion, the stomach is speedily relieved, and the vomiting does not continually recur without apparent cause, as in cyclic or persistent vomiting.

Cyclic vomiting is also very commonly diagnosticated as acute gastric catarrh, but in the latter disease the heightened temperature, coated tongue, pain, and tenderness in the epigastrium will, after the first twenty-four hours, allow us to differentiate the two diseases.

PROGNOSIS.—The prognosis varies according to the age of the individual affected. In young infants, especially in those whose vitality is weak, it may prove to be a very serious disease, from the exhaustion which invariably arises in the first twenty-four hours. The rule is that the younger the individual the more prostrating and serious is the disease. Even older children are at times so prostrated by the continuous vomiting that grave doubts are often entertained as to their ultimate recovery. In general, however, the prognosis in these cases is good, and I have never seen the disease result in death.

TREATMENT.—The treatment is essentially starvation during the first twenty-four hours. The child should be kept perfectly quiet in a darkened room. If after twenty-four hours the vomiting still continues, or even before, if there appears to be much exhaustion, or if the child is restless and sleepless and has an intermittent pulse, hydrate of chloral and bromide of potassium, dissolved in brandy and water, should be given by the rectum. These are intended to procure sleep and to stimulate the nervous centres. As a rule, however, the child is quiet, and sleeps in the intervals of vomiting, and, as the disease usually attacks an infant or a child who has been perfectly well, cardiac weakness is not commonly shown in the first forty-eight hours. No food and no drugs should be given by the mouth. After forty-eight hours, small enemata of peptonized milk can be given, and when the disease appears to have run its course, as it often does in three or four days, small quantities of a carefully modified alkaline milk can be tried cautiously by the mouth. A mistake is usually made in the treatment of the disease in feeding by the mouth too early.

The following cases are illustrative of this disease:

An infant, eight months old, strong and healthy, had always been fed on the milk of a wet-nurse. Without any previous symptoms he began to vomit, and continued to vomit every fifteen minutes for twelve hours. The intervals then became longer, and the vomiting ceased entirely on the third day of the attack. During the attack he emaciated rapidly, so that he seemed to be in the last stages of some wasting disease. He lay perfectly quiet and slept in the intervals of the vomiting. His mind was clear. His temperature was subnormal, and his pulse weak and intermittent. He was treated by rectal enemata of brandy, peptonized milk, and bromide of potassium.

He had several attacks of this kind in each of the following years of his life until he was five or six years old, when he would sometimes go for six months or a year without an attack. As he grew older the attacks became less severe, and when he was ten years old they ceased entirely.

Another case was that of a girl twenty-two months old, who was perfectly well until the vomiting began. Her pulse was slightly accelerated at first but gradually became slow and intermittent. Her temperature was normal. During the first four days of the attack the vomiting was almost continuous, and she became so weak and exhausted on the fourth day that it was feared she might die suddenly. There were great restlessness, dilated pupils, throwing of the head backward, slow pulse, and normal respirations. The emaciation was rapid. The urine was scanty. On the fifth day, the vomiting having continued, she fell into a state of collapse, the pulse was hardly perceptible, her countenance was ghastly and her extremities were cold. At one time after a severe attack of vomiting she became cyanotic, and was almost stifled by tenacious mucus. Some of this mucus entered the larynx during the attacks of vomiting, so that it seems as though her life was saved a number of times by the prompt action of an experienced nurse. On the sixth day the vomiting grew less, and on the seventh day it ceased. She was not, however, able to be up and about until the eleventh day, and was not fully well until the third week from the time that she was attacked. The treatment in this case was the same as in the one just described.

The third case was that of a boy nine years old. He was attacked suddenly with vomiting as described in the previous cases. The duration of the attack was about two weeks. The prostration was extreme and the boy's strength was supported solely by enemata, as at no time during the two weeks could anything be retained by the stomach.

These last two cases were unusually protracted.

GASTRALGIA.

In certain children, usually of a nervous temperament or in a debilitated condition, attacks of pain, seemingly referred to the epigastrium, seem to occur which are not accounted for by the more common sorts of pain in this region, such as in cases of vertebral caries and in indigestion, from which the pain of gastralgia is to be differentiated by the rules laid down in speaking of these other diseases. It is possible also that diaphragmatic pleurisy may cause a pain of this kind, and it must therefore be borne in mind in the diagnosis. These attacks are apparently neuralgic and produce symptoms of acute gastric indigestion, and, in severe cases, of obstruction. They should be treated during the acute attack with rest in bed, hot applications to the abdomen, deprivation of food by the mouth, and in extreme cases with a dose of tinctura opii camphorata 1 to 1.2 c.c. (8 to 10 minims). Between the attacks the general health should be improved by tonics and good hygiene. As malaria may be a source of the attacks, the plasmodium should be looked for, and if necessary the case should be treated with quinine, and later, when necessary, with arsenic in the form of Fowler's solution.

ACUTE GASTRIC INDIGESTION (Acute Dyspepsia).

By indigestion we mean a disturbance of the gastric secretions interfering with the function of the stomach to such a degree as to cause morbid symptoms. Exactly what this disturbance is in infants and young children has not been clearly proved. The cause of acute indigestion in infants, and in almost every case in young children, is the food which is given to them. This is especially noticeable in the first year. The ages in which indigestion most frequently occurs in this period are, first, in the early days of life, when the equilibrium of the breast-milk has not been established; second, in the middle of the first year, when the breast-milk is so apt to be replaced or supplemented by some other food; and, third, at the end of the year, when entirely new articles of diet are usually given to the infant.

Symptoms.—The symptoms of acute indigestion are extreme pallor, nausea, eructations of gas, a general appearance of discomfort, due probably to the pain induced by the development of gas in the stomach, with its resulting distention, and finally vomiting. If the diet is exclusively of milk, the vomitus will usually contain large curds of the coagulated proteids. In connection with the gastric disturbance there is commonly constipation, although sometimes there may be a relaxed condition of the bowels. The fæcal discharges accompanying these attacks are of an abnormal color, usually a mixture of green, white, and yellow, and of sour odor. There is little or no fever. At times the symptoms are so severe that the infant looks as though it were going to die. In rare cases, also, reflex symptoms of a serious aspect may arise, such as have been described under asthma dyspepticum (page 946).

Diagnosis.—Sometimes the diagnosis is obscured by the absence of vomiting, but the pallor and nausea are usually of sufficient prominence to allow us to decide that the seat of the disturbance is in the stomach. The differential diagnosis is most important between this condition and acute gastric catarrh. Acute indigestion is much more common, its duration is shorter, there is less fever, tenderness is unusual, and the response to treatment is much more rapid and evident.

PROGNOSIS.—The prognosis of acute gastric indigestion is in most cases good, but in infants whose vitality is extremely low, or in those that are debilitated by some wasting disease, these attacks are of serious import, and may prove fatal.

TREATMENT.—The treatment of acute gastric indigestion is to empty the stomach, either by washing it out or by the administration of warm water. A mild laxative should be given in order to clear away the undigested food, and the diet should be regulated. The laxative may be one or two teaspoonfuls of castor oil, .007 to .006 gramme (one-eighth to one-tenth grain) of calomel for four or five doses, or a teaspoonful of liquid magnesia. If the food has been human milk an analysis of the milk should be made at once, and the milk should be modified according to the rules which have already been given. If the infant is being fed on an improperly modified milk, or if improper food of any kind has been given to it, a recurrence of the attacks can easily be obviated by a modification of the elements of the food which seem to have produced the disturbance. Thus, in a number of cases I have found that whenever the infant's food was modified so as to raise the percentage of the sugar above 5, acute indigestion followed. In like manner in certain cases the percentage of the fat had to be reduced to 3, or perhaps 2.5, and the proteids even as low as 0.45, for a number of weeks until the digestive function of the stomach became normal. As relapses are common where food in any form is given too soon after the attack, nothing should be given excepting boiled water with a few drops of brandy for six or eight hours.

In older children the symptoms are similar to those which have just been described, and the diagnosis and treatment the same as in the infant. After allowing the stomach to rest, a recurrence of the attack is best prevented by at once placing the child for several days on an exclusive diet of a milk modified in such a way as to contain a percentage of from 2 to 3 of fat, 5 to 6 of sugar, 1 to 2 of proteids, and 10 of lime-water.

CHRONIC GASTRIC INDIGESTION (Chronic Dyspepsia).

If the attacks of acute indigestion are allowed to occur frequently from lack of proper treatment, a subacute or chronic form of the disease develops.

SYMPTOMS.—In infants the symptoms of chronic indigestion are much less severe than those of the acute form. The infant is apt to vomit after taking its food, to be restless, fretful, and either to lose in weight or not to gain. Its sleep will be very much disturbed, apparently by pain from flatus. In chronic indigestion the bowels are apt to be constipated, but this is not always the case. The chronic indigestion of older children presents a somewhat different aspect. The temperature is at times somewhat heightened. The tongue is apt to be coated, and the breath to have an odor. These children do not vomit so frequently as do infants. They lose in weight, become fretful, and get tired easily.

TREATMENT.—I have seldom found the use of any especial drug to be of much benefit in these cases of chronic indigestion. In quite a number of cases of both acute and chronic indigestion, before any food is introduced into the stomach it is often wise first to wash out the stomach thoroughly (lavage). This procedure is especially indicated if the indigestion has produced continuous vomiting.

The technique of washing out the stomach is very simple. A soft rubber catheter with a double eye, No. 21 French scale, for infants under six months, and No. 25 for older children, is attached by means of a piece of glass tubing 7.5 cm. (3 inches) long to another rubber tube which is 50.5 cm. (20 inches) long attached to a funnel, preferably of hard rubber, and capable of holding from 90 to 120 c.c. (3 or 4 ounces). The infant is seated upright in the nurse's lap, with its head inclined forward and resting on the nurse's arm. Its arms are controlled by a towel pinned around them. The catheter, having been wet with warm water, is easily passed over the base of the tongue into the stomach. As there is often considerable gas in the stomach, the funnel should be raised as high as possible above the infant's head, in order that the gas may pass out from the stomach. From 90 to 120 c.c. (3 or 4 ounces) of sterilized water should be poured into the stomach by means of the funnel. The funnel is then depressed below the level of the stomach, and the gastric contents will in this way be siphoned out. As the curds are often too large to pass through the eye of the catheter, a number of washings will often be necessary to break them up. By washing out the stomach not only are the irritating substances which are producing the indigestion removed, and the mucous lining of the stomach left free to recover its normal condition, but it is also possible to have a chemical examination of the contents made. Clinically, however, the latter is not necessary, although it is of great interest physiologically. No food should be introduced into the stomach for at least two hours after the washing. The washing of the stomach is almost entirely free from danger, and, in addition to being an important part of the treatment of indigestion, is often of great use where poisonous substances have been swallowed.

This method of treating disturbances of the stomach is more valuable in young infants than in older children, because the latter resist so vigorously that the remedy is often of more harm than good. The tube can, however, usually, even in older children, be introduced by aid of the ordinary gag which is used for intubation. Two assistants are usually necessary in introducing the tube in older children, while in infants one assistant is sufficient. In some cases it is found necessary to introduce the tube through the nose. The tube should be passed into the throat rapidly, since the gagging and vomiting occur chiefly when the tube touches the pharynx. There is usually an escape of gas or gastric contents as soon as the tube enters the stomach.

When the inflow of water through the tube is shown to be too rapid, by the fact that the infant holds its breath too long, or by its crying, vomiting, or coughing continuously, the flow should be stopped for a short time. Care must also be taken not to introduce the catheter too far into the stomach, as it may bend on itself and interfere with the flow of the returning water and gastric contents. If the gastric contents are expelled along the side of the tube rather than through it, the tube should be withdrawn until the vomiting has ceased. There seems to be no danger of passing the tube into the larynx, or of perforating the stomach with it.

Lavage is contra-indicated when there is cardiac disease or any severe pulmonary disturbance, and when the introduction of the catheter continues to excite vomiting it should be used with extreme caution. The fact that the infant is in a feeble condition is not a contra-indication to this treatment.

In connection with lavage it is well to speak of forced feeding (gavage) in the treatment of infants and young children. In cases of acute and chronic indigestion, and also where a catarrhal condition of the stomach is present, the infants at times refuse to take any food whatever. This does not occur merely when the disturbance is in the stomach; I have frequently met with it in severe cases of all kinds of disease. In a number of instances, when the infants would probably have died of starvation had not gavage been employed, this means of providing for their nourishment has been very successful. Forced feeding may sometimes have to be employed for a number of days, and even weeks, before the child will of itself swallow again.

The technique of gavage is similar to that of lavage. The same apparatus is employed, and the child should be placed flat on its back in bed or held half reclining. The head is held by an assistant. The catheter should be passed into the stomach rapidly, the funnel raised up in the air for a few minutes in order that the gas may escape, and the amount of food adapted to the age of the child should then be poured into the funnel. As the last of the food disappears from the funnel, the catheter is pinched tightly and quickly withdrawn. This precaution is important, in order that the pharynx shall not be irritated either by the slow withdrawal of the catheter or by the trickling of the remains of the fluid, as vomiting may in this way be excited.

One of the advantages which has resulted from the use of the stomach-tube is the knowledge we have acquired of the time which the food remains in the stomach at different ages. Thus, it has been found that during the early weeks of life the stomach is nearly emptied in an hour, while in older infants two hours are required for the same process. This knowledge is especially valuable when we are regulating the intervals of feeding in premature infants, and in infants during the first six months of life.

When other means can be employed, they are preferable to the stomach-tube, but in many instances when infants or children refuse to take their food the simplest way of forcing it upon them is to pinion the arms with a towel and have the nurse hold the child half reclining in her lap. Sometimes an assistant is needed to hold the head, but this is often unnecessary. Simply pressing the child's nostrils with the thumb and finger will cause it to open its mouth, and the food can then be poured in with a spoon, or introduced by the Breck feeder or by means of a dropper with a large end.

A child two and one-half years old, who has recently been under my care, for several weeks would not take any food without being forced to do so. Although this child was very ill with pneumonia, involving both lungs, it was fed every two or three hours, night and day, by this method. After the first two or three feedings it did not resist, and the nose did not have to be pinched, all that was necessary being to threaten to do so. 120 c.c. (4 ounces) of milk were, after a little practice, introduced by means of the dropper into the child's stomach in five or six minutes.

I have found that the most speedy cure of chronic indigestion is to give the child a carefully modified alkaline milk. In some cases it will be necessary to reduce the fat or sugar, in others the proteids, but in every case, as soon as it is determined which of these elements in full strength does not suit the individual digestion, an improvement in the symptoms will soon follow the reduction of the percentage of that element. In some cases peptonization of the milk after modification is found valuable. After the indigestion has been relieved by this means, other articles of diet adapted to the age of the child can gradually be added.

In addition to the direct treatment of the stomach, the intestinal disturbance which almost always accompanies the gastric indigestion should be relieved by occasionally giving a dose of some mild laxative, preferably one of the salts of magnesia, such as the citrate. This latter treatment is indicated not only for children, but for young infants, because, when there is gastric indigestion, the undigested food which passes into the duodenum is a prolific source of intestinal disturbance. This, by adding to the discomfort of the child, weakens it, and tends to prolong the gastric indigestion. In the more intractable cases, small doses of bismuth, soda, or dilute hydrochloric acid may be useful. Bismuth is indicated for allaying irritation and counteracting fermentation. Soda and hydrochloric acid have to be used somewhat empirically, as we know so little about the chemical conditions present. After the more prominent gastric symptoms have disappeared, considerable benefit can be obtained by using small doses of the tincture of nux vomica for several weeks.

ELIMINATIVE.

In the eliminative class of diseases of the stomach, certain morbid and irritating substances are supposed to enter the stomach, as though it were an excretory organ, and may possibly explain many of the obscure gastric symptoms which arise in early life, but at present our knowledge of this class of cases is so slight and indefinite that it need only be alluded to.

III. ORGANIC.—The organic affections of the stomach may be divided into *non-inflammatory* and *inflammatory*. They are very rare in comparison with the functional diseases.

A. NON-INFLAMMATORY.—The non-inflammatory conditions of the stomach comprise a *diminution in the size of the organ*, *mechanical dilatation*, *ulcers*, and *new growths*.

CONTRACTION OF THE STOMACH.

In certain cases the gastric capacity of the stomach is decidedly diminished. This diminution in size, as a rule, depends upon a lack of use, such as occurs in infantile atrophy. Sufficient food to fill the stomach is not taken, and in this way the stomach is not called upon to perform its normal work. In cases, also, in which there is continuous vomiting, this same lack of use may produce a diminution in the size of the stomach. These cases are of pathological rather than of clinical interest, as they can seldom be diagnosticated, and their treatment is essentially that of the especial disease to which they are secondary.

DILATATION OF THE STOMACH.

A moderate dilatation of the stomach is rather more common in infancy than in older children. The higher degrees of dilatation are rare. It may rarely arise from some malformation, such as a stenosis of the pylorus, but in most cases is the result of errors in feeding. It is more apt to occur when the infant is not nursed, unless especial care is taken to give the infant the quantity of food which is adapted to its age and gastric capacity. When the infant is nursed, the breast seems to provide the amount of food which is suitable. Dilatation from errors in feeding may be caused by the fact that the food is not adapted, either in quality or in quantity, to the age of the individual infant. When the quality is at fault, the nutrition of the tissues of the stomach is interfered with, and its walls become weak, and are thus more easily distended by the gas which results from the abnormal changes in the food. In this way dilatation occurs. This class of cases is notably represented in the disease rhachitis, in which dilatation of the stomach takes place very readily.

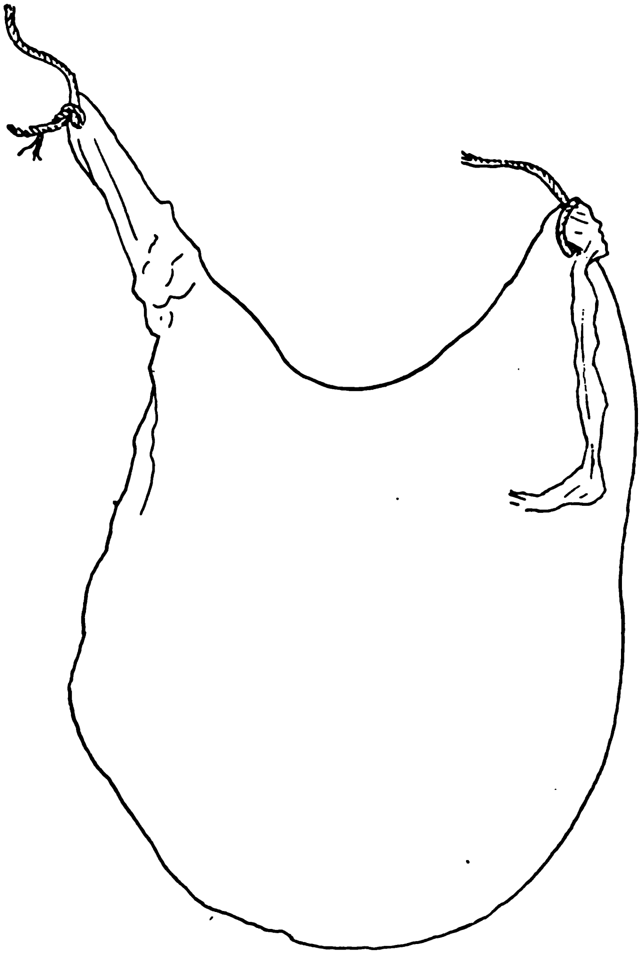
When the quantity of the food is not properly adapted to the size of the stomach, dilatation can take place in even a healthy infant, so that the careful regulation of the amount of food which is given at each feeding during the first year of life is most important.

PATHOLOGY.—The pathological condition which exists in cases of gastric dilatation is well represented in Fig. 161.

This stomach was taken from an artificially fed rhachitic infant, seven months old, who died under my care at the Boston City Hospital. The gastric capacity in this case was 300 c.c. (10 ounces), which corresponds to the gastric capacity of an infant twelve months old. The shape of the stomach is very significant of the symptoms.

The lesser curvature is not much altered, while the greater curvature is very much increased. The pathological condition of the tissues in these cases is such as would be expected from general malnutrition. In such diseases as rhachitis there is a stretching of the muscular fibres, as well as an atrophied condition of the entire gastric walls.

FIG. 161.



Dilated stomach. Rhachitic infant, 7 months old (Actual size.)

SYMPTOMS.—The symptoms of dilatation of the stomach are essentially those of chronic indigestion. Vomiting is quite frequent, and continues until the stomach has been entirely emptied, when a period of relief comes, to last until fresh irritation arises from another supply of food. Abdominal pain, flatulence, and general discomfort are prominent symptoms. Rapid loss in weight and emaciation also occur. In some cases, in young infants, convulsions may arise, apparently due to the reflex disturbance which is produced. There are usually considerable thirst and loss of appetite. When the dilatation is of a high grade, the vomiting may occur only after considerable intervals,—twenty-four to forty-eight hours,—during which time the food does not pass out through the pyloric orifice to any degree, but collects in the stomach.

Under normal conditions the stomach is somewhat tubular in shape and oblique in position. The food thus easily passes through the cardiac to the pyloric orifice. In dilatation of the stomach, on the contrary, the greater curvature is so much increased and depressed below the level of the pyloric orifice that a pouch is formed. The food, collecting in this pouch as though it were at the bottom of a well, has to be practically pumped, by the contraction of the muscular walls, up to and through the pyloric orifice. The already weakened stomach thus has to perform work for which it is not fitted, and finally is relieved by spasmodic vomiting. When only the small amount of food adapted to their normal gastric capacity is given to young infants whose stomachs are dilated, a large space of empty stomach is left above the level of the liquid which has entered the stomach. This creates a feeling of emptiness and general discomfort, so that the infant appears to be hungry when, in fact, it is only suffering from the feeling of incomplete filling of the stomach.

On inspection the abdomen is seen to be distended and tense, and on percussion to be highly tympanitic in its upper part. Succussion is not an especially valuable diagnostic sign in dilatation of the stomach. Succussion is so frequently found in many conditions, and is so likely to be confounded with that which occurs in the colon, that it cannot be relied upon. The outlines of a normal stomach when somewhat distended vary so much in infancy that the results of percussion are often very misleading. When, however, the tympanitic resonance is found to extend below the line of the umbilicus, we may suspect that we are dealing with gastric dilatation, but even then it is uncertain whether it is the gastric tympany which we have obtained. In infancy the cardiac end of the stomach is so slightly developed that any great increase in the area of gastric resonance to the left is an important aid in making the diagnosis.

DIAGNOSIS.—The differential diagnosis is to be made chiefly from dilatation of the colon. In many cases when the colon is dilated it is impossible to determine whether the stomach is also dilated, since under these circumstances the colon can almost completely cover a largely dilated stomach. In cases in which the diagnosis is uncertain, a valuable means of determining the presence of dilatation is artificial distention. This can be done without harm or much discomfort to the child by filling the stomach, several hours after the last feeding, by gas generated by giving part of a seidlitz powder in divided portions, by air through a stomach-tube, or preferably by water.

The gastro-diaphane, recommended by Koplik, can also be used as a method of diagnosis.

PROGNOSIS.—If the dilatation is due to congenital stenosis of the pylorus the prognosis is very unfavorable. In other cases the prognosis depends upon whether the condition arises from improper amounts of food or from some disease, such as rhachitis. In the former class the prognosis is good, and the stomach under a proper regulation of the diet soon resumes its natural size. In the second class it is not so good, and, as a rule, the stomach will remain more or less distended until the disease which causes the dilatation has been cured.

TREATMENT.—If the case is an obstinate one, lavage is an important part of the treatment. In many cases, however, good results are obtained simply by regulating the quality and quantity of the food. When the food is first given in the proper amount it will not fill the stomach nor satisfy the demands of the infant. Under these circumstances the infant will be very restless, and will often cry almost continuously from the time of one feeding until the next. The nurse must be made to understand that these signs of discomfort are liable to last for a number of days, until the stomach has more nearly resumed its normal size, and that an additional supply of food must not be given to the infant because it cries for more.

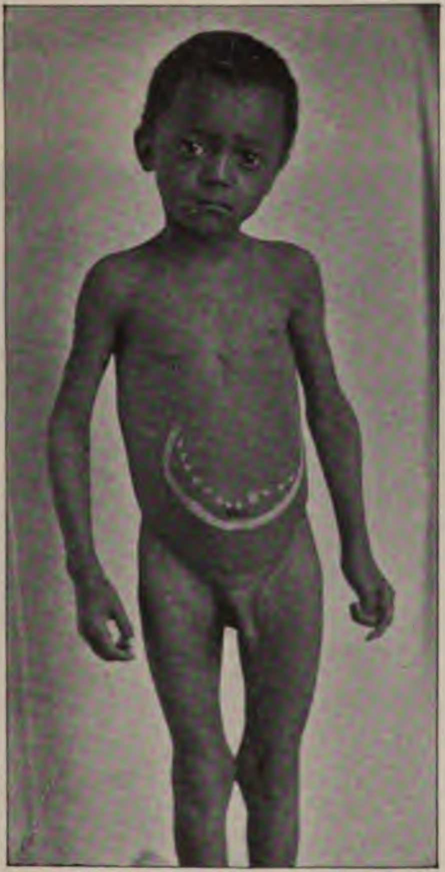
The following case illustrates dilatation of the stomach as it occurs in the first year of life. A male infant four months old was well and strong at birth. It was not nursed, but was fed on a mixture of milk, cream, and water. It was unusually vigorous, and is reported to have never been satisfied with the small quantities of food suitable to its age. When it was three weeks old it was given 150 to 180 c.c. (5 or 6 ounces) at each meal. Somewhat later, in its second and third months, it gradually developed symptoms of indigestion, and when I was called to see it was in a very serious condition. It had been having frequent and prolonged convulsions. At times when it was in the convulsions it would fall into a state of collapse, the pallor of its face would be extreme, and it would look as though it were dying. On examination nothing abnormal was found in the thorax. The entire abdomen was found to be distended, especially in the upper part, where the gastric tympany was pronounced and easily marked out by percussion. The percussion showed the stomach to be dilated, and to extend below the line of the umbilicus and far to the left of the median line.

The infant was given small amounts of food at frequent intervals. For the first two or three days it cried and screamed for more food, but the convulsions ceased, its general condition improved, by the end of the week the distention of the stomach had subsided very markedly and the infant had become tranquil, and this time there was no recurrence of the symptoms.

A second case was that of a colored boy six years old. He was markedly rhachitic. He was reported to have been in fair health, though delicate, until one month previous to entering the hospital, when he began to have persistent vomiting. He had lost greatly in weight, had been very restless at night, and he had continual borborygmi.

Physical examination showed marked abdominal enlargement. On percussion the gastric tympany was found to extend downward as far as the umbilicus, 7.8 cm. (3 inches) to the right of the median line, and 10.4 cm. (4 inches) to the left. In the figure the line of percussion which represents the greater curvature of the stomach is marked with white dots. As the resonance of the colon was also exaggerated in this case, and as its differentiation from that of the stomach was somewhat difficult because it evidently overlapped the lower border of the stomach, an endeavor was made to eliminate this obstacle to diagnosis by mechanical means. The child was given one-half a seidlitz powder dissolved in water. The other half, which had also been dissolved in water, was next swallowed. The child showed no signs of discomfort, and stated that he did not feel any pain or any more tenderness in the epigastrium than before the powders were taken.

FIG. 162.



Dilatation of stomach. Age, 6 years.

The outline of the distended stomach could be fairly well seen, and on percussion the line of the greater curvature was found to be 2.5 cm. (1 inch) below the line of the umbilicus, the colon having been pushed out of the way by the distended stomach. I have indicated the line of greater curvature by a broad white line.

(Subsequent history.) In this case it was not found necessary to wash out the stomach more than once or twice, for as soon as small amounts of food were given at frequent intervals the vomiting ceased and the stomach gradually resumed its normal size. At the end of two months the child left the hospital free from any abnormal gastric symptoms.

ULCERS.

Ulcers of the stomach in infancy and early childhood are very rare, but cases have been reported. They may be *non-inflammatory* or *inflammatory*, the distinction between the two often being very difficult to make. They may occur as a result of hemorrhage into the tissues of the stomach.

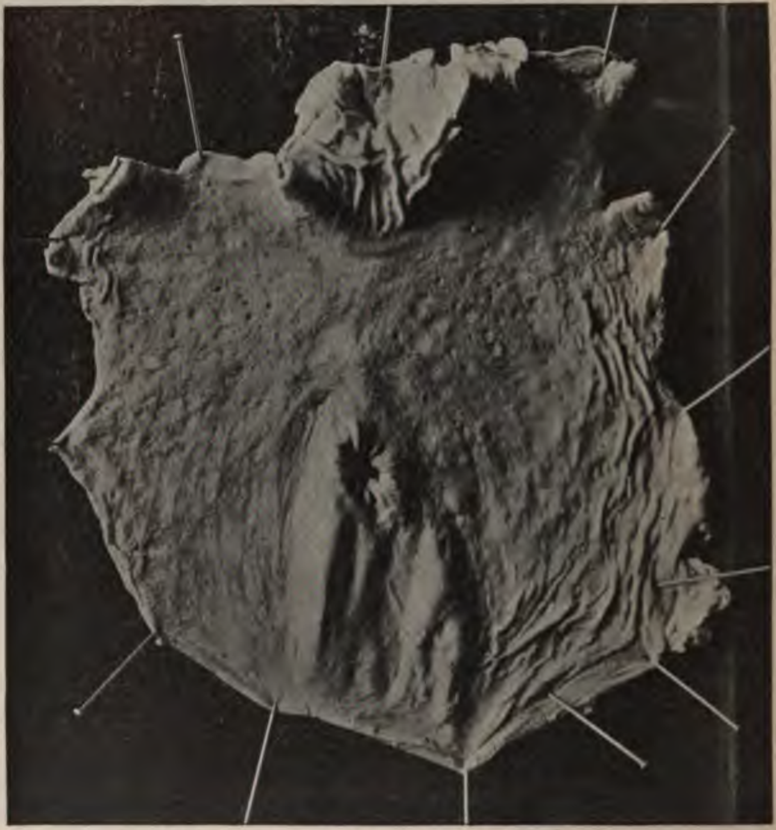
They may be of *follicular* or *tubercular* origin, and the *round perforating* ulcer has also been found.

There are no diagnostic symptoms, but when vomiting of blood occurs ulceration may be suspected.

The prognosis is bad. The treatment is necessarily symptomatic.

Fig. 163 represents the stomach of a female infant one year old under the care of Dr. Northrup.

FIG. 163.



Follicular ulceration of stomach. Female, 1 year old.

The infant was under treatment for one month. It had vomiting and diarrhœa. During the first week that it was in the hospital its temperature varied from 38.8° to 39.4° C. (102° to 103° F.), after that being normal or subnormal. The respirations varied from 40 to 50, and the pulse from 120 to 140. In the second week it began to refuse its food and to emaciate. The diarrhœa continued and the vomiting was persistent. The vomitus was somewhat brownish in color, and the child died of exhaustion.

On examining the stomach it was found that the lining mucous membrane was covered with small ulcers, varying in size from dots to 1 cm. ($\frac{3}{8}$ of an inch) in diameter. The lesions appeared to be follicular ulcerations. In the middle of the specimen was a much larger ulcer which perforated the posterior wall. There was no evidence of an inflammatory condition, and the cause of these lesions was unknown. There was, however, a certain degree of necrosis around the ulceration.

NEW GROWTHS.

Morbid growths in the stomachs of infants and young children are so extremely rare that their occurrence need merely be referred to.

B. INFLAMMATORY.—The inflammatory lesions of the stomach may be either acute or chronic, and are termed *gastritis.*

ACUTE GASTRITIS.

Acute gastritis may be divided into (1) *gastritis catarrhalis*, (2) *gastritis corrosiva*, and (3) *gastritis pseudo-membranosa*. The cases in which organic lesions of the stomach can be proved to exist are very limited in comparison with those in which the functional disorders are present. In a large number of cases which are spoken of as gastritis catarrhalis no catarrhal condition is present, and they would be much better classified under the heading of indigestion. Cases are numerous in which a diagnosis of gastritis has been made during life and in which at autopsy no definite lesion has been found. When, however, gastritis is present, as a rule the acute form is more common in infants, while the chronic form occurs more frequently in children towards the age of puberty.

Gastritis Catarrhalis Acuta (Acute Gastric Catarrh).—The cause of acute gastric catarrh is somewhat obscure, but it is usually supposed to arise from an exaggerated form of indigestion, or from the presence of irritants of various kinds. Infection may also be a cause, especially in the membranous form.

PATHOLOGY.—The pathological lesions which characterize acute gastric catarrh are hyperæmia of the mucous membrane, hypersecretion of mucus, small punctate hemorrhages, and slight thickening of the mucous coat. In certain cases the inflammation has been found especially to affect the follicles. According to Booker, when a catarrhal condition of the gastric mucous membrane is present, the milk remains much longer in the stomach than under normal conditions, possibly four or five hours, or even more. A microscopic examination of the gastric contents in these cases shows various micro-organisms, and sometimes epithelioid and pus cells. The small number of bacteria found in cover-slip preparations from the contents of the stomach affords a most striking contrast to the large number of bacteria which under like circumstances are found in the fæces.

Two forms of acute gastric catarrh are usually described, the division being made according to the severity of the disease into *subacute* and *acute.*

SYMPTOMS, SUBACUTE.—In the subacute form there is little or no fever, while in the acute the temperature is high. The subacute form is by far the more common. It is difficult and almost impossible to state definitely the symptoms of this form of gastric catarrh, as they so nearly approach to those which occur in cases of indigestion in which we believe no gross pathological conditions exist, that we should always be guarded in our use of the word catarrh. Pain is so common a symptom in all gastric disturbances, the existence of tenderness is so difficult to determine in infants and young children, and a hypersecretion of mucus is so often known to occur without the presence of an inflammatory condition, that there does not seem to be any one symptom on which we can rely. A general picture of the disease which is supposed to represent this form of gastric catarrh is that of slight fever, nausea, vomiting of food mixed with mucus, and at times of mucus alone, with a sense of tenderness, uneasiness, and discomfort in the epigastrium. There may be frontal headache, a rather swollen, coated tongue of somewhat glossy appearance, and often a slight follicular pharyngitis. There is loss of appetite, with at times hiccough and eructations of gas. The bowels are usually constipated at first, but after three or four days diarrhœa may be present. When an infant or child seems prostrated for a few days, and sick beyond what would be expected in an acute attack of indigestion, and when in combination with a slightly heightened temperature there is frequent vomiting of mucus, we are justified in supposing that we are dealing with a subacute gastric catarrh.

SYMPTOMS, ACUTE.—Acute gastric catarrh is rare, but is a much more serious disease than the subacute form. It is characterized by high fever, 39.4°, 40°, 40.5° C. (103°, 104°, 105° F.). The invasion is sudden. The disease may last for two or three weeks and show severe and alarming symptoms. There may be active vomiting, delirium, and sopor in the beginning, so that it may be impossible to determine whether or not one of the other acute febrile diseases is developing. The characteristic symptoms of gastric catarrh develop later, and then the differential diagnosis is easily made.

Instead of the cessation of the vomiting in the first twenty-four or forty-eight hours, as in scarlet fever, and of the continuance of the cerebral symptoms, as in meningitis, or of the development of pulmonary symptoms, as in pneumonia, the vomiting continues, though not quite so frequently as in the beginning, the mind becomes clear, and the symptoms point to the abdomen rather than to the head or thorax. The onset of pneumonia in some cases, though in my experience rarely, simulates this disease. The pulse is rather irregular. There is usually constipation at first, followed by diarrhœa later.

PROGNOSIS OF SUBACUTE AND ACUTE GASTRIC CATARRH.—The prognosis in both these forms of catarrh is good except in very debilitated children.

TREATMENT.—The treatment of cases of subacute and acute gastric catarrh is the same as that described in speaking of indigestion. Food should be withheld from the stomach for many hours, for it remains so long in the stomach that a fresh supply at short intervals will act as an additional source of irritation. In those cases which do not respond readily to long intervals of rest and to feeding with small quantities of a modified alkaline milk, lavage will prove of value. Much judgment should be used as to the time when the food is to be increased in strength, for unless great precautions are taken relapses will frequently occur, and as a result the disease may finally become chronic. After convalescence has been established the child will begin to gain in weight. Some simple tonic, such as nux vomica, is usually indicated for a week or ten days until the child has recovered its strength. During the beginning of the attack, when food is being withheld, if the child is made very restless by extreme thirst, teaspoonful doses of iced soda-water can be given, but with caution and as seldom as possible. After the irritation has been somewhat allayed, it is found valuable in certain cases to give dilute solutions of brandy, barley water, or white of egg in the form of albumin water before beginning with the treatment just spoken of. Albumin water is best made by dissolving the white of an egg 20 to 30 c.c. ($\frac{2}{3}$ to 1 ounce) in water 120 c.c. (4 ounces), and heating to 40° C. (104° F.). In this way the albumin is held in solution, and the albumin water can be put on ice until needed for use. In especially intractable cases nothing should be put in the stomach for some time, nutriment being given in the form of peptonized milk with enemata.

In the acute form, besides the treatment just mentioned, the child should be placed in a darkened room, soothing applications applied to the abdomen, and small quantities of iced soda-water given. If there is much exhaustion, stimulants are indicated.

Gastritis Corrosiva Acuta.—Corrosive lesions of the mucous membrane of the stomach are at times produced by swallowing irritants, such as arsenic, carbolic acid, and caustic fluids. In these cases the lesions are usually found on the summits of the rugæ.

Treatment.—The treatment is by washing out the stomach with large quantities of water, administering the proper antidote, and feeding the child on a liquid diet so modified as to be as little irritating as possible to the injured mucous membrane.

Gastritis Pseudo-membranosa.—The membranous form of gastritis is extremely rare in infancy and childhood. Cases have been reported, notably those of Wollstein. In these cases the congestion of the rugæ was very marked, and along the greater curvature extended over an area of a number of inches. There was a thick grayish-green membrane, with some erosions. The gastric walls were much thickened.

Symptoms.—The symptoms of gastric disturbance in these cases are often almost entirely absent, but there may be vomiting, pain, and tenderness in the epigastric region, and insatiable thirst. A pathognomonic symptom would be the vomiting of shreds of membrane, with or without an admixture of blood. This symptom is, however, extremely rare, because the membrane is usually adherent, so that a differential diagnosis is often impossible.

Prognosis.—The prognosis is very unfavorable.

Treatment.—The treatment is purely symptomatic.

CHRONIC GASTRITIS.

Chronic gastritis is not usually met with in infancy, but occurs in later childhood, and is then in form catarrhal. It is especially common in the summer months, and is generally the result of neglect or of improper treatment of the acute form of the disease.

Gastritis Catarrhalis Chronica (*chronic gastric catarrh*).—PATHOLOGY.—The pathological condition which is found in chronic gastric catarrh is the result of long-continued hyperæmia. There is often a slaty discoloration of the mucous membrane, with cellular infiltration of the submucosa. In addition to this there is usually found a considerable quantity of tough mucus.

SYMPTOMS.—The symptoms are not so clearly defined as in the acute form of the disease, but are variable and of a rather sluggish type. The tongue is apt to be much coated and the breath to have a disagreeable odor. There is considerable abdominal distention after meals, so that the children complain that their clothes feel uncomfortable.

Frontal headache is apt to occur. The children gradually grow thin and anæmic. They vomit at irregular intervals, and are usually constipated. There is often a slight cough, and the symptoms, so far as the stomach is concerned, may form so small a part of the general picture of the disease that the child is not infrequently brought to the physician on account of its cough and because it is supposed to have some pulmonary affection.

PROGNOSIS.—Although the disease is often somewhat intractable, the prognosis under proper treatment is good. It may last for three or four months; but in many cases which are usually considered chronic gastric catarrh it has seemed to me there is no organic lesion, but that the disease is functional in its character, and the prognosis consequently very good.

TREATMENT.—It is often necessary in these cases to precede the treatment by carefully washing out the stomach. We must remember, however, that a considerable quantity of mucus may be in the stomach, which cannot be removed by washing, so that if the symptoms continue after one or two washings, even though no mucus is returned by the tube, we should repeat this treatment from time to time. The diet should be an alkaline modified milk, with a low percentage of proteids, if necessary peptonized, and a moderate percentage of fat and sugar. The percentages of the different elements should be increased as improvement in the gastric symptoms takes place, and later broths and milk can be tried. Symptomatically in certain cases pepsin, dilute hydrochloric acid, and bismuth are occasionally indicated. A valuable tonic in the after-treatment of these cases is nux vomica.

DISEASES OF THE INTESTINE.

GENERAL CONSIDERATIONS.—Diarrhœa.—As vomiting is the most significant symptom of gastric disturbance, so diarrhœa resulting from increased intestinal peristalsis is the most characteristic symptom of intestinal disturbance. Diarrhœa is always a symptom, never a disease. There seems to be a predisposition to diarrhœa in the first two years of life, which decidedly lessens as the child grows older. The most frequent time for the occurrence of diarrhœa is during the summer months.

Prophylaxis.—Much can be done at all seasons of the year to prevent the occurrence of diarrhœa, but prophylaxis is of the utmost importance in warm weather. The children should be protected by proper clothing from extremes of heat and cold, and from dampness. They should, if possible, be taken away from crowded or unclean districts in cities and towns during the hot weather, and have the advantages of fresh country or sea air and good hygienic surroundings. Both the quality and the quantity of the food should be carefully regulated, care being taken not to give too much food, as the depression due to heat weakens the digestive function. In very hot weather an extra amount of water should be allowed and the solid food somewhat diminished in amount. As milk is the most common source of infection in the diarrhœal diseases, and as bacteria multiply more rapidly as the weather becomes warmer, it is especially important to have the milk fresh and sterile during the summer months. It must be understood that not only bacteria but also their toxines, which are not destroyed by pasteurization or sterilization, may produce most serious results. Uncooked fruits and food are contra-indicated in very hot weather. Especial attention should be paid to any slight indisposition which may arise in hot weather, as it may render the child more vulnerable to the various causes of diarrhœa.

Intestinal Contents.—The intestinal contents should be studied in regard to their color, consistency, composition, odor, and amount. The changes which take place in the intestinal contents are significant of diseased conditions, but not necessarily of any especial disease.

COLOR.—The color of the fæcal discharges varies greatly, and it would be impossible to describe completely all the variations from the normal when there is disturbance of the intestinal contents. A number of the abnormal colors more usually seen are shown in Plate III., facing page 84.

The square numbered 16 is what is usually spoken of as clay-colored. This clay color may be due to a diminution in the amount of bile which enters the intestine, or to undigested fat. This color is abnormal, and is usually met with in intestinal diseases of a subacute or chronic type. It does not necessarily indicate a serious condition, however, as even a small plug of mucus may interfere with the flow of bile into the duodenum.

Number 17 may be simply a change that has taken place after the fæces have been passed, and which often is not significant of any especial pathological condition. It may, however, show that the changes which have taken place in the food during its passage through the intestine have not been entirely normal. It is the least important of the changes which take place in the color of the intestinal contents. The colors in 18 and 19, are what may be seen in a more serious disturbance of the enteric tract. These colors may appear in any of the intestinal diseases which are accompanied by diarrhœa, but are significant of no especial disease. They are merely to be considered pathological in contradistinction to the normal colors seen in 3, 4, 6, 7, 8, 9, and the beginning abnormal condition represented in 17.

Besides these shades of green there are a great many varieties of color produced by the mixture of green, yellow, white, and brown. These are valuable merely as instructing us whether we are dealing with a normal or an abnormal condition of the intestinal contents, and are not significant of any one disease, either functional or organic. Much variety in the color also arises from the admixture of blood, mucus, and shreds of membrane. In this connection it is well to remember that the yellowish-white lumps seen in undigested fæces are often made up of fat as well as of proteid material.

The color of the intestinal contents may also be changed by the administration of various drugs, such as iron, which causes a more or less black color. Bismuth gives the colors which are seen in 12, 13, and 14. Number 12 is the color which was produced by giving to an infant 0.18 gramme (3 grains) of bismuth every two hours for six doses; number 13, when 0.24 gramme (4 grains) of bismuth was given every two hours for six doses; and number 14, when the latter dose had been omitted for twenty-four hours. The size of the dose and the intervals between its administration will of course produce different shades of color.

When the solids of the intestinal contents are much reduced in proportion to the serum, as in cases of acute and frequent diarrhœa, the discharges become more and more fluid, and sometimes almost entirely lose their color and look like water.

Consistency.—In the first year of life, or while the infant is having only milk for its food, the consistency of the fæcal discharges is intermediate between solid and fluid, and the discharge, as a rule, is smooth and free from lumps. As the infant begins to take other forms of food and a mixed diet, the fæcal discharges gradually become more solid. The consistency of the fæcal discharge is abnormal when it becomes liquid, as in diarrhœa, or when it is too solid, as in constipation.

Composition.—In addition to the various substances which make up the food which enters the intestine, the fæcal discharges contain bile, mucus, epithelial remains, and many bacteria. In diseased conditions they may also contain certain morbid elements, such as blood, pus, and membrane. In intestinal diseases of both an acute and a chronic type the mucus may be very largely increased, but it cannot be considered to be especially characteristic of an inflammatory condition, as the secretion of mucus apparently may be very much increased in purely functional conditions. The bacteria are very numerous and of many varieties, but in most cases the detection of any especial form of these organisms does not aid us in diagnosticating the especial disease. Notable exceptions to this statement are where one finds the typhoid bacillus, the comma bacillus, and the amœba coli.

ODOR.—While in the normal fæcal discharges of infants fed entirely on milk the odor is comparatively slight, it becomes much stronger as other articles of food, either of a starchy or of a proteid nature, are added to it. When an abnormal condition exists, various changes take place, as in acid fermentation, in which the odor is supposed to be sour, and in albuminous decomposition, in which the odor is supposed to be very foul, but these distinctions cannot with our present knowledge be said to be definitely proved. Although these can scarcely as yet be considered of great diagnostic importance, they are sufficiently so for us to make use of them in the diagnosis and treatment of intestinal diseases. Thus, when acid fermentation is supposed to be present, a reduction in the percentage of the sugar is indicated, while when albuminous decomposition is suspected a reduction of the proteids in the food is called for.

AMOUNT.—In estimating the amount of the fæcal discharges we must consider the total amount in twenty-four hours, and not the large or small amount which may occur at one movement. The total amount in twenty-four hours is of much importance in both the acute and the chronic diseases of the intestine. In the acute diseases, the more frequent the diarrhœa and the larger the amount the greater is the exhaustion and the worse is the prognosis. In some chronic diseases the total amount of fæcal discharges may be very large. In these cases the larger the total amount the less has been the absorption and the worse is the prognosis, for this condition is an indication that the child is being starved from a lack of power to absorb the food which has been given to it.

Diseases of the intestine may be divided into three classes,—*developmental*, *functional*, and *organic*.

I. DEVELOPMENTAL.—Certain malformations and malpositions of the intestines occur as a result of abnormal development. The malformations have already been considered under diseases of the new-born. Malpositions are met with in infants when there is a transposition of the abdominal organs, or when portions of the intestines are found to be outside of the abdominal cavity.

II. FUNCTIONAL.—The functional diseases of the intestine may be classed as *acute*, *chronic*, and *eliminative*. The acute functional diseases of the intestine may arise from *nervous* disturbance or from *indigestion*, which is sometimes more marked in the duodenum, and sometimes in the intestine beyond the duodenum.

ACUTE NERVOUS.

In certain infants and children whose nervous system is easily affected exaggerated peristalsis causing diarrhœa may arise from a number of causes without any known lesions, fever, or gastric disease. Among these causes may be cited heat, cold, and fright. In like manner in these individuals foreign bodies, food or otherwise, may by simple reflex irritation cause such a nervous disturbance as to produce diarrhœa. In these cases either the small or the large intestine, or both, may be affected, and, so far as we know, the mucous membrane is either normal or simply hyperæmic. There is more or less serous exudation. These cases are rare in comparison with the other forms of diarrhœa, such as those which are caused by bacteria, and in them intestinal decomposition and intestinal inflammation are not present primarily.

Symptoms.—The symptoms of simple diarrhœa are very apt to appear suddenly. There is usually abdominal pain, not, as a rule, of great intensity. At first there are two or three rather liquid yellowish-brown discharges occurring at intervals of perhaps one-half or one hour, and often accompanied by considerable flatus. There is a certain amount of restlessness, pallor, and exhaustion. Vomiting is rarely excessive. The temperature, as a rule, is not raised, or is raised very slightly. The pulse is rather weak and somewhat accelerated. The number of the discharges may be eight, ten, or twelve in the twenty-four hours, and these soon become watery and of a lighter color, but are seldom green. The odor is somewhat increased, but not excessively. These symptoms, unless they are exaggerated by improper food or by bad treatment, usually disappear in a few days.

Treatment.—The treatment is essentially to remove the cause. If the cause is atmospheric, a change in climate is sometimes necessary. If undigested food is causing intestinal irritation, a dose of castor oil, 3.75–7.50 (drachms 1 to 2), or calomel, .006 gramme ($\frac{1}{10}$ grain), every hour for ten doses, may be given, but, as a rule, this is not necessary. The child should be kept in bed. Food should be withheld for some hours. After the cause has been removed, the treatment should be to control the peristalsis and the pain. For this purpose a few drops of tinctura opii camphorata and hot applications to the abdomen are indicated. If there is a tendency for the diarrhœa to continue, the subnitrate of bismuth may be given, and in some cases when there are signs of exhaustion a stimulant may be needed. In the cases in which the excessive peristalsis seems to be prolonged by weakness, brandy in small doses is more useful than astringents or opium. For several days the diet should be simply milk heated for twenty minutes at 75° C. (167° F.), and containing ten per cent. of lime-water. These simple diarrhœas, especially in hot weather, should never be allowed to continue, as they render the intestine vulnerable to the more serious diseases caused by micro-organisms, which may at any moment gain an entrance in this way.

ACUTE INDIGESTION.

Disturbances arising from intestinal indigestion, although in most cases having their origin in the duodenum, seem also in quite a number of cases to be located in the intestine beyond the duodenum. It has yet to be proved that any pathological lesion is present in these cases, and for the present they must be looked upon as functional.

These cases vary in their symptoms with the individual and according to the part of the intestine which is most involved. In one set of cases where the irritation is probably below the duodenum, the disturbance of digestion is shown simply by the increased peristalsis, such as has been described in the nervous cases, but here the evidence points to an undigested condition of improper articles of food which have been given and which appear in the discharges. The treatment of this class of cases is very simple, and consists in first giving a laxative and then regulating the diet according to the age of the individual.

Symptoms.—What is usually spoken of as a "bilious attack" represents another class of cases. This condition is very rare in infancy, and usually occurs in middle and later childhood. In these cases, in addition to the increased peristalsis and evidence of undigested food, there are frequently icterus and vomiting of bile, indicating that there is irritation in the duodenum. In addition to these symptoms there may be headache and excessive nausea. The icterus is usually slight in degree, but often is marked and is noticeable in the conjunctivæ and in the urine. Plate III. (facing page 84), No. 11, shows the staining of bile on the napkin from the urine of an infant during an acute attack of indigestion involving the duodenum and accompanied by icterus. The temperature in these cases is usually slightly raised for a few days and then becomes subnormal. In a certain number of cases the fæcal discharges become clay-colored. This color is often produced mechanically, as has already been explained.

Prognosis.—Although the symptoms in these acute attacks may often appear quite serious, the prognosis is always good. In some individuals they are liable to recur even when the diet and the general health are well attended to.

Treatment.—In the treatment of this class of duodenal disturbances we must consider that the fats in the food are in all probability especially liable to prolong the disease by not being properly digested so long as the function of the duodenum is involved. We should, therefore, in treating these cases, lessen the amount of fat given in the food. I have found that the treatment which most speedily shortens the attack is (1) total restriction from food for twelve hours, with the administration of small quantities of cold, sterilized water if the thirst is excessive, and (2) the administration of small quantities of milk modified as in the following prescription:

PRESCRIPTION 86.

Fat	0.16
Sugar	6.00
Proteids	3.00
Lime-water	10.00

The mixture to be heated to 75° C. (167° F.) for twenty minutes; from 120 to 180 c.c. (from 4 to 6 ounces), according to the age, to be given every three hours. In some cases also the diet of proteids may be varied by giving expressed beef-juice or solutions of white of egg.

Under this treatment the icterus usually passes away in a few days, and the child can then soon be given its ordinary food.

CHRONIC.—The chronic functional diseases of the intestine may be the result of acute nervous disturbances, or they may arise from a number of prolonged attacks of acute indigestion. They may be divided into (*a*) *nervous*, (*b*) *tubular*, (*c*) *duodenal indigestion*, (*d*) *intestinal indigestion*, (*e*) *incontinence*, (*f*) *constipation*.

CHRONIC NERVOUS.

In the chronic form of nervous functional intestinal disturbance either the small or the large intestine may be affected, and the condition of the mucous membrane, so far as we know, is either normal or hyperæmic. The causes are the same as in the acute form. This class of cases is not especially common, as they are merely a prolongation of the symptoms which I have already sufficiently described in speaking of the acute cases. The treatment of these cases is essentially with stimulants and care of the general health.

TUBULAR.

In addition to these more common intestinal affections of nervous origin is one that is called *tubular*. This disease is so rare before the age of puberty that it need only be alluded to. It is a condition of the mucous membrane of any part of the intestine in which an exudation of mucus takes place in such a way that masses closely simulating a membrane may occur on the surface. When discharged through the rectum they are sometimes found to have formed a cast of the intestine. These masses are mostly made up of mucus, and may occur in shreds of greater or less extent, as well as in the tubular form. The disease is supposed to be of nervous origin.

SYMPTOMS.—The symptoms are pain, tenderness, and tenesmus. The temperature is usually normal.

PROGNOSIS.—The prognosis, as a rule, is good, although in some cases the disease may be much prolonged.

TREATMENT.—The treatment is to be directed towards the improvement of the general health and the nervous condition, the local treatment being simply symptomatic.

CHRONIC DUODENAL INDIGESTION.

The chronic form of functional intestinal indigestion plays an important part in intestinal disease, especially when it is located in the duodenum. Chronic indigestion of the duodenum constitutes a disease of itself, and is one of the most difficult to cure. It has usually been spoken of under the names of chronic gastritis, duodenal catarrh, and mucous disease. We at present, however, have no proof that catarrhal or any other pathological lesions of the mucous membrane are present in these cases, and the weight of evidence is in favor of the view that the disease is purely functional.

ETIOLOGY.—The etiology of chronic duodenal indigestion is in many cases obscure, but in a large number of cases it is produced by the continued administration of food which is not adapted to the age or digestive capacity of the child. It is at times met with as a sequela of some exhausting disease, such as typhoid fever, pneumonia, pertussis, or one of the acute exanthemata. It very rarely occurs in early infancy, being commonly met with during the middle and later periods of childhood.

SYMPTOMS.—The symptoms are at first somewhat varied. The disease may be preceded by a number of attacks of gastro-enteric indigestion of a subacute character. A tendency to nausea and vomiting extending over a number of months may sometimes precede the full development of the disease. The gastric disturbance, however, is not marked, and is probably a reflex condition depending upon the functional disturbance of the duodenum. At first the fæcal discharges show merely the various changes which occur in ordinary indigestion, sometimes manifesting a tendency to diarrhœa and sometimes to constipation. The color of the discharges at this early period is not significant of anything beyond ordinary indigestion, and is usually a mixture of yellow, white, and green. As the disease progresses, certain characteristic symptoms arise and definitely mark its presence. Mucus begins to appear in the fæcal discharges, and soon becomes quite large in amount. It may occur in shreds or masses of considerable size, and is sometimes found covering hard lumps of fæces.

With this hypersecretion of mucus, which is not necessarily an indication of an inflammatory condition, the child begins to be fretful, to be wakeful at night, to grind its teeth, and to lose in weight. The skin becomes dry, and there is usually a coexisting follicular pharyngitis which causes a short, dry cough. The child gets tired easily, and complains of pain in the epigastrium after eating. The abdomen is apt to be distended and tympanitic. There are frequently frontal headache, a coated tongue, and a disagreeable odor to the breath. The fæcal movements now begin to become clay-colored, and the skin to assume a sallow tint, with at times a slight amount of icterus. Sometimes an exacerbation of all the symptoms takes place, resulting in an acute attack of indigestion. These symptoms, varying in intensity, and sometimes ceasing to be prominent for days or weeks, usually continue for months, and in intractable cases may last for years. The temperature in this disease is usually normal, sometimes subnormal, but may of course, where an exacerbation occurs, be somewhat raised. The pulse is usually moderately slow. Sometimes a subacute form of bronchial catarrh accompanies the disease, but it does not appear to be a part of it. The appetite varies, is often unnaturally great, and there is sometimes a craving for large quantities of sugar.

Diagnosis.—When all the symptoms are present, the diagnosis of chronic duodenal indigestion is not difficult. The appearance of the child is characteristic. Its eyes are dull and heavy; its skin is dry and harsh and sometimes slightly icteric, while the loss of flesh, the distended and tympanitic abdomen, and the coated tongue are more marked than in any other disease. When, in addition to this picture, an examination of the fæcal discharges shows them to be clay-colored and to contain a large amount of mucus, the diagnosis is quite evident. The disease which is most commonly mistaken for chronic duodenal indigestion is pulmonary tuberculosis. The short, dry cough, the emaciation, and in some cases the bronchial catarrh, often make parents and physician fear that this serious disease is present. If, however, the entire history of the case is studied carefully, pulmonary tuberculosis can soon be eliminated.

Prognosis.—The prognosis of chronic duodenal indigestion is in most cases good. Even in those cases which last for a period of years the health is usually entirely restored. When, however, the disease has lasted for a long time, and the child is in a very debilitated condition, the prognosis becomes more serious.

Treatment.—The treatment of this disease is essentially by diet, and not by drugs. Such articles of food should be given as will be chiefly digested by the stomach and will not tax the duodenal digestion. This of course indicates a proteid diet, and contra-indicates the administration of starches, sugars, and fats. In order not to tax the disturbed duodenum by overloading it in its weak condition, small amounts of food at shorter intervals than usual are found to produce a better result than the regular three or four daily meals. The diet which I have found most valuable in treating these cases is a milk so modified as to have a low percentage of sugar and fat, a high percentage of proteids, and ten or fifteen per cent. of lime-water. Soups of various kinds, and meat, can also be given, and the crust of bread in limited quantity. A valuable adjuvant to this treatment, as a mild astringent and stimulant, is a small amount of claret, preferably given in seltzer water. The meals should be five in the twenty-four hours. It is exceedingly difficult in most cases to keep the child on this diet, but if it is rigorously enforced the duration of the disease will be decidedly shortened. As the epigastric pain and the amount of mucus in the discharges grow less, the diet may be somewhat varied by giving fish and eggs, and the percentage of lime-water in the milk may be reduced to five. As relapses occur very easily, however, it is generally best to continue with this rigid diet until the fæcal discharges have become normal in color and have not shown the presence of mucus for a number of weeks. In mild cases when there is much constipation, small doses of calomel, or any mild laxative, are indicated. Podophyllin can also be given, as in this prescription (Prescription 87):

PRESCRIPTION 87.

Metric.	Grammes.	*Apothecary.*	
℞ Podophyllin	0.06	℞ Podophyllin	gr. i;
Alcohol	3.75	Alcohol	ʒi.
M.		M.	

Sig.—From 3 to 5 drops, according to the age of the child, in the morning and evening, lessening the dose if it causes more than two discharges daily.

When there is a tendency to diarrhœa, small doses of bismuth are found to be valuable.

Tincture of nux vomica freely diluted in water and given in doses of a few drops after each meal seems in some cases to be of value.

The remainder of the treatment is essentially symptomatic, and if the children are weak and anæmic tartrate of iron and potassium can be given.

During the whole course of this disease cod-liver oil is contra-indicated, but when the disease has been cured and the child is left weak and emaciated it may in some cases be beneficial. Its administration, however, should always be carefully supervised, as it may cause a relapse. The specially restricted diet should be continued for some time after the characteristic symptoms of the disease have disappeared, or relapses will occur.

The following case is illustrative of chronic indigestion of the duodenum. A girl six years old and healthy at birth, was nursed until she was thirteen months old. During her first year she had an attack of bronchitis. Previous to this attack she had never had any intestinal disturbance. Her abdomen was always rather prominent. From her earliest infancy she had been a nervous child, had not slept well, had talked much in her sleep, and had occasionally walked in her sleep. During the first six months of the disease the symptoms were chiefly loss in weight and cough. She then had an attack characterized by vomiting for twenty-four hours, followed by anorexia, fever, languor, and apathy; the bowels became constipated the skin icteric, the urine dark-colored, and the fæcal movements light-colored. She had an intense craving for sugar, and ate all that she could lay her hands on, so that she had to be watched very closely to prevent her satisfying this morbid appetite. Her appetite for other articles of food was poor. The abdomen was distended and tympanitic, and the tongue coated. The breath had a disagreeable odor, and there was loss of flesh. There was a follicular pharyngitis, which evidently caused the cough.

The child was placed on the following diet. Her first meal was milk so modified as to contain fat 2, sugar 3, proteïds 4, lime-water 10. With this meal she was allowed to have a small amount of the crust of French bread. The second meal consisted of broth and the crust of French bread, and one ounce of claret in half a tumbler of seltzer water. The third meal consisted of meat, the crust of French bread, claret, and seltzer water; the fourth, of soup, the crust of French bread, claret, and seltzer water; the fifth of the modified milk and the crust of French bread. After each meal three drops of tincture of nux vomica were administered.

This diet was carried out rigorously for one week. At the end of that time the child looked much better, the urine was clear, the fæcal movements began to resume a more natural color, the mucus in the discharges was very much lessened, and the abdomen was not so much distended. The tongue was less coated, and the cough had almost disappeared. It was also found that the craving for sugar had much decreased. The diet was then slightly increased in variety. At the end of a month the child had recovered entirely, and some weeks later an ordinary diet at the usual times was given to her.

CHRONIC INTESTINAL INDIGESTION.

There is a class of cases in which the disturbance seems to be located in the intestine beyond the duodenum and to be comparatively free from gastric symptoms. These cases are characterized by their chronic course, by the irregular character of the intestinal discharges, which at times are normal, and by presenting when abnormal most varied appearances.

Symptoms.—As a rule, these infants do not gain regularly or normally. They become anæmic. The skin may be dry. They are fretful, do not sleep well, and in fact present the usual signs of malnutrition.

Treatment.—The treatment is to study carefully each case and feed it according to its especial idiosyncrasy. No especial drugs are indicated.

INCONTINENCE OF FÆCES.

Incontinence of fæces is a condition in which the fæcal movements occur involuntarily. This may be due to inability to control the sphincter ani, or to loss of power of the sphincter itself. The first class of cases, due to disease or injury of the spinal cord, or to absence of reflex sensation, is rare. The second class is due to an atonic condition of the sphincter. The treatment of the first class of cases is essentially that of the disease in which it occurs. The treatment of the second class is illustrated in the following cases:

A boy, eleven years old, much overworked at school, had been made to study a number of languages, and allowed to take only a very limited amount of exercise in the open air. He completely lost control of the sphincter ani, and became very anæmic and weak. Nothing abnormal was found on a physical examination. The boy was taken from school, relieved entirely of his studies, and kept in the open air most of the day. Under this treatment, in addition to the administration of tartrate of iron and potassium and claret, he improved rapidly, and in two months was entirely well.

Another boy, seven years old, came to the Children's Hospital, with a history of incontinence of fæces lasting over a year. He illustrated the condition of incontinence from habitual constipation, as the incontinence was found to depend on the stretching of the rectum by impacted fæces. The rectum was emptied by a dose of castor oil and an enema each day. At the end of a week the boy had ceased to have involuntary fæcal movements, and has since continued well.

CONSTIPATION.

By constipation is meant a condition in which the movements of the bowels do not take place as often as is normal for the individual, and in which the consistency is abnormally increased. Constipation is a symptom and not a disease. It is a relative term, as what would be normal in one individual may be abnormal in another. During the first year of life two or three daily discharges may be considered normal, in the second year two discharges, and in the third and fourth years one discharge is the usual number. The causes of constipation are varied and, in many cases, rather obscure. They may be mechanical, but are usually functional, and either *spasmodic* or *atonic*. As the sigmoid flexure is proportionately long in infancy, flexions may occur with the result of producing a mechanical obstruction.

Spasmodic.—The spasmodic cases are rare, but should be recognized, as they frequently cause much disturbance of the child's general health. They are usually due to fissures about the anus or to an increased size and consistency of the fæces, which, by causing pain in defecation, result in spasmodic closure of the sphincter.

TREATMENT.—The treatment of the *spasmodic* cases, when the cause is from fissure or, as in some instances, from hemorrhoids, is described on page 813. When an abnormal size or consistency of the fæces is producing the spasm, the rectum should be thoroughly emptied for a number of days by mechanical means. This may be in the form of enemata of equal parts of glycerin and water, or in extreme cases by using the finger, well oiled, to break up and draw out the pieces. The subsequent treatment would be the same as described below in the atonic cases. The size of the fæcal masses may at times be enormous, and in these cases a mechanical dilatation of the rectum may result. Gradual dilatation of the sphincter with bougies two or three times a week has proved a valuable adjunct to the treatment of these more severe cases, the rectum gradually recovering its normal caliber.

Atonic.—The atonic is the most common form of constipation, and simply represents a sluggish condition of the intestinal peristalsis. This may arise from weakness of the intestinal muscles. A weakened condition may result from a child not being taught to have its bowels moved at regular times. The lack of exercise which is so common in the child's life during the winter months accounts for many cases in which during the summer months, when the child is out of doors playing all day, the bowels are no longer constipated. Any disease of a debilitating nature, as well as the indiscriminate use of laxative drugs may cause this weakness of the intestinal muscles. The most common of all the causes, however, is the food, which is insufficient in amount or improper in quality for the digestion of the especial child. In infants who are being fed exclusively on milk, a low percentage of fat in the milk seems in a number of cases to produce constipation. I have noticed, however, that when a breast-milk contains a low percentage of fat and a high percentage of proteids the fæcal movements are feeble and watery. In some cases boiling, or even pasteurizing, the milk produces obstinate constipation, but in many cases no such condition is produced. In young children a lack of the proper amount and combinations of vegetables, meats, and fruits is a very common cause of constipation.

SYMPTOMS.—In addition to the usual symptoms of infrequency of defecation, straining, and increased consistency of the fæces, there are others which are often overlooked. There may be general nervous symptoms, such as fretfulness, apathy, insomnia, and pain varying in its locality, sometimes being prominent in the head and sometimes in the abdomen. It is not uncommon to find streaks of blood with the fæces when the strain is excessive.

DIAGNOSIS.—The diagnosis of the condition of constipation is self-evident; the difficulty arises in detecting the cause and locating the part of the intestine in which the atonic condition exists. When it is the rectum that is chiefly affected there are, as a rule, no especial symptoms which cannot be relieved by the use of enemata or suppositories; when it is the upper part of the small intestines the symptoms connected with intestinal indigestion aid us in the diagnosis.

PROGNOSIS.—As a rule, constipation can be easily cured, but some cases are extremely intractable and last for a number of years. In the mechanical cases, as soon as the intestine has become more developed and assumed the relative proportions found in adult life, the constipation is very apt to pass away; so in these cases we may give a favorable prognosis.

TREATMENT.—The treatment of the *atonic* cases is to remove, when possible, the causes which have already been cited, and to give the proper treatment to the disease from which the constipation has resulted. Regular habits as to the movement of the bowels, exercise, and meals are very important.

Diet.—Reliance should be placed on the regulation of the diet, rather than on drugs, which should, as a rule, be looked upon as of only temporary use. When the infant is being fed on modified milk, we should remember that a variation in the percentage of the sugar, as well as of the fat, may produce constipation in certain individuals. During the first year the percentages to be obtained as soon as possible are:

Fat	4.00
Sugar	7.00
Proteids	2.00 to 3.00

During the second year whole cow's milk, with the addition of cream, should be given. It is often advisable to reduce the amount of cereals and increase the amount and variety of vegetables and fruits. Of the latter, baked apples, orange-juice, stewed prunes, and ripe peaches are especially indicated. Broths and meat-juices should also be given, and as the child grows older, sometimes at two years, butter, and at two and a half years meat, should be added to the diet. Hot or cold water given before breakfast, either with or without orange-juice, is often efficacious. Massage of the abdomen in certain cases of obstinate constipation has been a most successful form of treatment. Suppositories of either gluten or glycerin are especially efficacious when the rectum is chiefly at fault. Enemata, either of soapsuds and water or equal parts of glycerin and water, are very useful in temporarily removing the symptoms until the more radical form of treatment by the regulation of the diet is thoroughly established.

Drugs.—When drugs are indicated, purgatives and, as a rule, laxatives should be given but sparingly, and only with the idea of giving temporary relief. In older children, cascara can be given, either in the form of the elixir, 3.75 to 7.50 c.c. (1 to 2 drachms), or the fluid extract, 0.18 to 0.3 c.c. (3 to 5 minims). Nux vomica, according to the age of the child, is especially valuable in the form of the tincture, 0.12 to 0.24 c.c. (2 to 4 minims). For young infants the milk of magnesia, 1.88 to 3.75 c.c. (½ to 1 drachm), or olive oil, 3.75 c.c. (1 drachm), or phosphate of soda, 0.12 c.c. (2 grains), should be given. All these drugs should be given in as small doses and as seldom as is sufficient to produce a satisfactory result, and should be omitted as soon as possible.

ELIMINATIVE.

Under the term eliminative disturbances of the intestine are included a number of unexplained and obscure symptoms which we at present are unable to classify elsewhere. It is probable that they will be more fully understood in the future. It seems as though the intestine often acts as an organ for the elimination of various morbid products from the economy. The diarrhœa which results from the irritation of these foreign elements is not distinguishable from that which occurs when the irritation is primarily in the intestine itself. Our knowledge of this class of disturbances is, however, so small that I shall merely refer to its possible occurrence.

III. ORGANIC.—The organic diseases of the intestine may be divided into *non-inflammatory* and *inflammatory*.

A. NON-INFLAMMATORY.—The non-inflammatory diseases of the intestine may be divided into *fermental*, *mechanical*, and *new growths*.

ACUTE FERMENTAL.

The non-inflammatory conditions of the intestine, which for want of a better term we speak of at present as fermental, include those which arise from acid fermentation and albuminous decomposition, which are produced by micro-organisms. The disturbances which arise from these causes represent the greater proportion of the diarrhœal diseases which occur during the warm months of the year.

ETIOLOGY AND PATHOLOGY.—The causes of fermental disturbance in the intestine lie in impure or improper foods and bad hygienic surroundings.

In both acid fermentation and albuminous decomposition it is probable that the small intestine is most affected. The condition of the mucous membrane may be normal, or there may be desquamative catarrh. The process may go no farther, or it may be followed by inflammatory changes in the intestinal mucous membrane.

The fermental class of cases holds a position midway between the nervous forms of intestinal disturbance and the inflammatory forms with their pronounced lesions.

Symptoms.—The great variety of causes which give rise to these fermental processes result in a variety of symptoms. The onset may be subacute, with little or no fever and without vomiting, or it may be acute and accompanied by a high temperature and active vomiting. After a variable period of general discomfort and restlessness, diarrhœa sets in, which varies so greatly as to its frequency, amount, color, and consistency that it would be impossible in the present state of our knowledge to divide these variations clinically. The onset of fermental diarrhœa is, however, so often characterized by the toxic symptoms of sudden rise of temperature, followed after a day or so by a normal temperature, that when we meet with this occurrence we are usually justified in eliminating the inflammatory and more serious intestinal lesions. In some cases the diarrhœa, although accompanied by much prostration and various nervous disturbances, disappears after a few days; in others, especially in the warm weather, it may last for a number of months. In this fermental diarrhœa the color of the discharges is commonly some shade of green or greenish yellow, and the odor is often very offensive, sometimes being the excessively sour one which is supposed to arise from acid fermentation, and at other times the extremely foul one of albuminous decomposition. The discharges are generally accompanied by considerable pain and a large amount of gas. They are usually not more than a dozen in number, and are larger in amount than those which occur in ileo-colitis. The symptoms are often so severe that the disease has a serious aspect, but in a considerable number of cases after the intestine has been thoroughly emptied the temperature falls and the nervous symptoms subside. In some of the more severe cases certain special symptoms become so prominent as almost to produce different types of the disease. The most important of these are excessive vomiting, continuous hyperpyrexia, and extreme restlessness with insomnia. There is usually rapid and great loss of weight. In cases which are not prolonged by fresh irritation or by unwise treatment recovery often takes place quite rapidly. According to Morse, albuminuria occurs in about fifteen per cent. of all cases. Casts, usually hyaline and fine granular, occur in about sixty per cent. of the cases of albuminuria. The renal condition in these cases is a degenerative one, and not a true nephritis.

Diagnosis.—When the attack is subacute, with slow onset, without vomiting, and with infrequent discharges, the diagnosis is not difficult, and is to be made from the nervous disturbances, which can usually soon be differentiated by the absence of fever and by rapid recovery. When, however, the onset is acute and is accompanied by vomiting, the diagnosis must often be held in abeyance, as the symptoms of high temperature, vomiting, and diarrhœa may be present in infants and young children in the initial stage of a number of acute diseases. The disease from which it is especially to be differentiated is cholera infantum. In fermental diarrhœa the prostration is much less, and the temperature after the early hours of the attack is much lower. The serous discharges and the continuous vomiting which soon arise in cholera infantum are quite different from the greenish discharges and the less frequent vomiting which occur in fermental diarrhœa. We must remember, however, that cholera infantum and the acute inflammatory intestinal diseases are usually preceded for a number of days by this fermental form of diarrhœa, and that the special micro-organisms which produce the former disease gain an entrance for themselves and their toxines by means of the abnormal intestinal conditions produced by the fermental changes. The differential diagnosis from ileo-colitis is considered under that disease, on page 822. It is also well to remember that gastro-enteric symptoms are often so pronounced during the early days of a pneumonia that they may mask the presence of that disease.

PROGNOSIS.—In previously healthy children the prognosis of fermental diarrhœa is good. It depends, however, upon the degree and the kind of fermental process which is causing the disease, and also on the amount of resistance to these processes which the individual possesses. It also depends upon the vulnerability of the individual to the other bacteria which may at any time complicate the disease, as in the more severe cases of the disease described above. The cases of infantile atrophy are especially liable to die when attacked by this as well as by any other form of intestinal disturbance. In these cases it seems as though the infant were totally unable to resist even a slight amount of toxic absorption. The prognosis, therefore, when an already debilitated child, or one with infantile atrophy, is attacked by fermental diarrhœa must always be guarded. It also depends upon how soon and in what way the disease is treated. The renal complications do not alter the prognosis.

TREATMENT.—The treatment of fermental diarrhœa is to remove at once the source of the disturbance by thoroughly emptying the intestine. When the vomiting is excessive it is sometimes necessary to wash out the stomach, but, as a rule, this procedure is not indicated. A dose of castor oil, one teaspoonful for infants under one year, and two teaspoonfuls for older children, is the best initial treatment. When the stomach is so sensitive that it does not seem advisable to give castor oil, 0.06 to 0.12 gramme (1 or 2 grains) of calomel can be given in divided doses. In the more severe cases, and when there is a tendency to a prolongation of the acute symptoms, irrigation of the intestine is indicated. Food should be withheld for a number of hours,—at least half a day, if possible. Stimulants are indicated when there is much prostration. The only other drug which in my experience seems to be needed is bismuth subnitrate, which should be given in large doses until the disease has run its course and the diarrhœa has ceased. The amount of bismuth in the severe cases should be from four to eight grammes (sixty to one hundred and twenty grains) in twenty-four hours.

Milk can in most cases be given after the first twelve to twenty-four hours if it is properly modified. It should contain from ten to fifteen per cent. of lime-water, and at first should have the percentages of all its elements considerably reduced. The milk which is used for this purpose must be fresh, since it is not sufficient to sterilize it, as the toxic products of bacteria may still be present in it and thus add fresh irritation to that which has already been produced by the fermentation. In many cases it is impossible in the present state of our knowledge to determine what special form of fermentation is present. When acid fermentation appears to be prominent, the milk should be so modified as to contain a low percentage of sugar, while when albuminous decomposition with its excessively foul odor is met with, the proteids should be reduced to a fraction. Whether this treatment will in the future be proved to be the best it is impossible to state, but on the ground that various forms of bacteria are the cause of these disturbances, and that the special form of bacteria which is producing them has been developed in the food on which it thrives best, it certainly seems reasonable, and should be adopted until further light is thrown upon the subject.

When breast-milk or fresh modified cow's milk cannot be obtained, weak animal broths, such as those made from mutton, chicken, or beef, can be used. Opium is almost invariably contra-indicated in these cases, and serious results may arise from its administration. The peristalsis which occurs as the result of fermental irritation is a conservative process of nature, intended to carry away the morbid products which have resulted from the fermentation. Under these conditions the administration of opium prevents the elimination of the poison from the intestine and allows it to remain and produce still further irritation, or to be absorbed and give rise to still graver septic symptoms. Towards the end of the attack, when the intestine has been thoroughly emptied, small doses of opium in the form of tinctura opii camphorata may be used with caution to diminish pain and to control the excessive peristalsis which may result from nervous exhaustion after the disease has run its course. In these cases, however, stimulants are more valuable than opium. Extreme and continuous hyperpyrexia should be treated by bathing in water heated to 32° C. (90° F.) and gradually reduced to 25.5° C. (80° F.). Excessive restlessness is to be treated with bromide of soda in doses of 0.012 to 0.006 gramme (2 to 10 grains). In some cases subcutaneous injections of morphia, beginning with 0.0006 gramme ($\frac{1}{100}$ grain), is indicated.

When a child in the warm weather has once had an attack of fermental diarrhœa it is very apt to have a number of attacks. Its diet, therefore, should be carefully regulated for a considerable period, and if possible it should be taken to the sea-shore or the country until the return of cool weather.

As especial illustrations of the great variety of fermental diarrhœas which are liable to be met with in warm weather the following cases may be taken as examples:

A child three years old and perfectly well was attacked suddenly with abdominal pain, nausea, pallor, and prostration. He vomited once or twice and was found to have a temperature of 40° C. (104° F.). Within a few hours he began to have frequent fæcal dejections of sour odor, lessened consistency, moderate amount, and a peculiar dark green color (Plate III., No. 18, facing page 84). This green is one of the more common colors met with in fermental diarrhœa. At first the discharges took place every hour, and later every three or four hours. After the first twenty-four hours the temperature became normal and in three or four days the diarrhœa ceased entirely.

The next case was that of an infant thirteen months old. On entering the hospital it was much emaciated and had a slight diarrhœa, caused apparently by improper food. Its temperature was only slightly raised. On examining it nothing else abnormal was detected. The diarrhœa was infrequent and was not accompanied by any other especial symptoms. It soon began to improve, gained in weight, and had a normal temperature. After it had been in the hospital one week it suddenly began to have diarrhœa characterized by large frequent discharges of lessened consistency, of foul odor, and of the color which is seen in Plate III., No. 19, facing page 84, which is a mixture of yellow and light and dark green. The discharge seemed from its foul odor to be an illustration of what is called albuminous decomposition. These colors are, however, only relative and are not diagnostic. In this acute attack the temperature was raised at first but soon fell to a little above normal. The infant lost greatly in weight, became extremely emaciated, and looked as if it would die. The skin often became cold, and the prostration was extreme. These symptoms continued for three or four days, and the number of the discharges in the twenty-four hours varied from seven to ten. The symptoms gradually became less severe and the diarrhœa abated. Later the diarrhœa stopped entirely, and the infant gained rapidly in weight and strength.

CHRONIC FERMENTAL.

The cases of acute fermental diarrhœa at times are prolonged for many weeks, and even months, and thus produce a chronic form of diarrhœa. This occurs especially in children who are the subjects of rhachitis, syphilis, and general tuberculosis, also in those with chronic broncho-pneumonia. The continuous administration of improper food may produce this condition, as may also improper exposure from insufficient clothing.

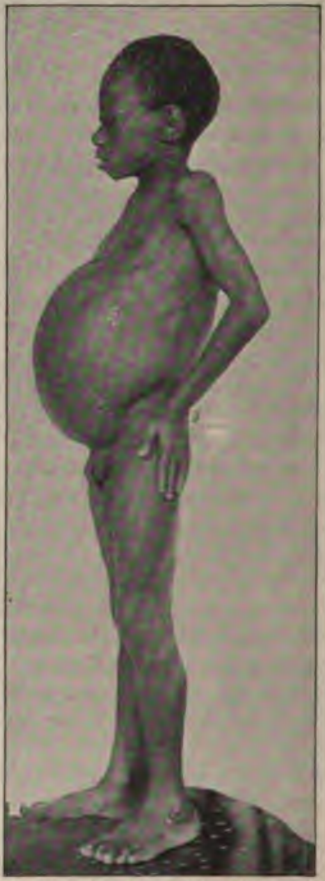
MECHANICAL.

The mechanical diseases of the intestine are quite numerous, but with few exceptions are not of special importance medically, and belong rather to the province of surgery.

DILATATION OF THE COLON.

In comparison with dilatation of the stomach, dilatation of the colon is very rare except as a temporary condition. It is liable to occur at any time from an over-production of gas. If permanent, it is usually congenital, or it may be secondary to some congenital malformation such stricture of the intestine. The chief symptoms are constipation and di tention of the abdomen. The condition is usually fatal, but adult li may be reached. The treatment is essentially surgical.

FIG. 164.



Dilatation of colon. Male, 12 years old.

The following case illustrates a dilatation of the colon, which was seeming caused by a congenital stricture, and in which an artificial anus was made by Halste The child recovered from the operation, but later, owing to still further obstructio he had to be operated on again and died. In this case the extreme distention of th abdomen, which was tympanitic through its whole extent, the evident obstruction the fæcal discharges, and the absence of symptoms pointing towards gastric diseas would suggest a dilatation of some part of the intestine, presumably of the colon.

VOLVULUS.

By volvulus is meant a twisting or bending of the intestine. This condition is more apt to occur in early life than later, possibly because of the greater proportionate length of the mesentery at this time, which allows the intestine greater latitude of motion. It occurs either by itself or in connection with intussusception, from which it is to be differentiated by the absence of blood and mucus in the discharges.

INTUSSUSCEPTION.

Intussusception or invagination is a condition in which a part of the intestine has passed down into another part. Under these circumstances there is an outer layer of intestine within which is the part of the intestine forming the invagination. Only a small portion of the intestine may be invaginated, or it may extend from the ileo-cæcal valve to the rectum. Small invaginations are frequently found at the post-mortem examinations of infants and young children. These probably take place during the death-struggle, as no pathological condition is found in connection with them. This form is usually multiple and in the small intestine. The form of intussusception which occurs during life is very rare under three months, and is most common from the third to the sixth month. At this age the large intestine is shorter in relation to the small intestine than in the adult, while the mesentery is relatively wider, and thus allows much greater latitude for misplacement, especially of the cæcum and colon. The etiology of intussusception is obscure, but it is probably directly due to increased local peristalsis.

The pathological condition depends upon the tightness of the constriction and the length of time from the beginning of the obstruction. In some cases the incarcerated portion of the intestine is so little constricted that the bowel remains pervious. In other cases the constriction is so great that the tension of the intestinal capillaries quickly becomes so extreme that hemorrhage occurs, and inflammation, with resulting adhesions, is apt to follow rapidly. The intestine may not only be invaginated, but may be bent on itself, an important point to remember in regard to treatment.

SYMPTOMS.—The symptoms of intussusception are usually more acute in infants than in older children. In infants they are often at first rather obscure. Paroxysmal pain and discharges of blood from the rectum occur. Later the blood is mixed with mucus and looks like currant jelly. There is usually vomiting, which may be stercoraceous. The mind is clear, and in young infants the face is often tranquil between the paroxysms of pain, so that on looking at the infant it would scarcely be supposed that a serious condition was present. Later, however, the face grows haggard and the eyes become sunken. During the first twenty-four to forty-eight hours, and even longer, the infants will often take their food quite readily. Tenesmus is at times present. There may be fever, especially when inflammation has occurred. The pulse is usually quickened. These symptoms all vary, and depend on the amount of the invagination. In some cases these are the only signs which indicate that there is abdominal disturbance. In many instances, however, either at once or within a few hours, a tumor can be felt in the abdomen.

Diagnosis.—The chief points in the diagnosis of intussusception are the occurrence of discharges of blood, vomiting, abdominal pain, and the detection of an abdominal tumor, usually on the left side of the abdomen. In these cases a careful rectal examination should always be made, for a tumor can often be found in this way when an external examination has failed to detect it.

Prognosis.—Without treatment the prognosis is unfavorable, though there are a certain number of recoveries by spontaneous reduction, or rarely by sloughing of the invaginated portion of the intestine, which is then passed by the rectum. If death takes place, it usually occurs about the third or fourth day, or at any rate within a week, after the incarceration is complete. When the incarceration is not complete the infant may live for many weeks, and in older children in rare instances the disease may become chronic.

Treatment.—The treatment of intussusception, when the diagnosis has been definitely made, should be immediate, as in no other disease does a delay result in more serious consequences. Food and cathartics or laxatives are contra-indicated. If the infant shows signs of collapse, small quantities of brandy-and-water should be given. In the early hours of the attack an attempt should be made to reduce the intussusception by hydrostatic pressure. This can be easily done by having the infant's buttock's somewhat raised and introducing water under a pressure of about one metre (3⅓ feet) by means of a fountain syringe. The water should be lukewarm, and should have dissolved in it salt in the proportion of one teaspoonful to a quart. The abdomen should be gently rubbed at the same time. In some cases this procedure results in a reduction of the intussusception, but when this is not accomplished an anæsthetic should be given and the attempt repeated.

Even when inflammation has not begun and adhesions have not formed, the pressure of the column of water may fail to reduce the intussusception, because the invaginated portion may be bent on itself, so that the hydrostatic pressure increases the obstruction rather than relieves it. When adhesions have taken place and when there is great congestion, as sometimes occurs during the first twenty-four hours of the attack, hydrostatic pressure is usually unsuccessful and may be dangerous. If this method has failed, the infant should be placed at once in the hands of a surgeon, as under these circumstances an early laparotomy will give the most favorable results.

The following case of intussusception illustrates the importance of not delaying operation.

A male infant, six months old, nursed by its mother, and previously perfectly healthy, after a slight loss of appetite for several days began to have abdominal pain in the morning, and in the middle of the day had a discharge of blood from the rectum unmixed with fæcal matter or mucus. The bowels had been thoroughly moved on the previous day, and there had been no tendency to constipation. During the afternoon there were five or six discharges of blood. In the evening the infant looked well and did not show any signs of discomfort except occasional slight attacks of abdominal pain and an indisposition to nurse. The rectal temperature was 39° C. (102.2° F.). An examination of the abdomen externally and by the rectum revealed nothing abnormal. The infant had a restless night, vomited several times after nursing, and had six discharges of blood. The temperature was 38.3° C. (101° F.), the pulse 135, strong and regular, and the general appearance good. The abdomen was soft and not tender on pressure, but towards the umbilicus, under the left costal border, a rather ill-defined cylindrical tumor could be detected.

Hydrostatic pressure was employed to reduce the intussusception, but failed. The surgeon who saw the infant on the second day decided to wait twenty-four hours before performing laparotomy. On the following day the infant died suddenly.

At the post-mortem examination nothing abnormal was found except an ileo-cæcal intussusception. An examination of the invagination showed that the retained cæcum was so twisted that the lower opening was directed to one side of the axis of the intestine, and the hydrostatic pressure from below must have simply packed the sac tighter and rendered reposition more difficult. The invagination involved 20 cm. (8 inches) of the intestine. The serous surfaces were firmly adherent through their whole extent, and considerable force was required to reduce the invagination without tearing it. The reduction, however, was successfully accomplished, the adhesions giving way and the intestine being left uninjured and apparently healthy. This case illustrates how necessary it is to employ the most skilled surgical aid in these cases.

HERNIA.

Hernia is essentially a surgical disease, and has been referred to in the division on diseases of the new-born.

FISSURES.

There are a number of lesions which occur about the anus in infants and young children which, though somewhat rare, should be recognized for purposes of differential diagnosis. They are, however, so purely surgical in their treatment that they need only be mentioned here.

One of these conditions is that of fissure, which occurs either at the anus or more commonly a little distance from the orifice. Pruritus and reflex urinary symptoms are common. Defecation is often painful, and constipation of the spasmodic type may thus result. A general nervous irritation at times results from fissures of the anus which may seriously interfere with the nutrition of the child. The treatment is cleanliness and the application of boracic acid ointment or the stick of nitrate of silver every two or three days. If these measures fail, stretching of the sphincter under anæsthesia usually cures the disease.

PROLAPSE.

Prolapse of the rectum is not uncommon in young children. It is usually produced by straining from various causes, especially in extreme constipation or in diarrhœa. The wall of the rectum comes down through the anus, and is easily recognized by the appearance of the mucous membrane. The prolapse is ordinarily transitory, but in the more severe forms the rectum remains down.

Treatment.—The treatment is to remove the cause. Constipation should be relieved first by enemata and then by keeping the movements of the bowels semi-liquid by means of gentle laxatives. The tenesmus accompanying diarrhœa may be relieved by sponging with ice-water or by the use of 0.015 gramme ($\frac{1}{4}$ grain) suppositories of cocaine. The child should be kept in bed for a number of days, the protrusion being gently pushed back each time that it comes down. The prolapse should be reduced with the fingers well oiled, the central portion being pushed back first. If the parts are very sensitive the reduction is facilitated by the application of a solution of cocaine. After reposition it should be kept in place by means of a pad and a T bandage. Under this treatment a large number of cases recover. The more serious and intractable cases, however, should be referred to a surgeon.

POLYPI.

Polypus of the rectum is more common in early life than at any other period. Hemorrhage from the rectum, when not due to constipation, diarrhœa, or fissure, usually arises from polypi. A careful examination for this growth should be made when rectal bleeding is frequent or large. Rectal polypi are of various sizes, and may be myxofibromata or adenomata. The surface of the polypus is usually smooth, and the pedicle is often long and thin. The diagnosis is easily made by a digital examination. The treatment is either to twist or cut off the polypus. The growth is not apt to recur.

HEMORRHOIDS.

Hemorrhoids are rarely met with in infancy or early childhood, but can occur and should be treated by the same methods as in later life.

FISTULÆ.

Fistulæ in ano is not a very common condition in infancy or early childhood, but is met with at times. The condition has the same characteristics as in the adult, and should be treated in the same way.

NEW GROWTHS.

New growths in the enteric tract are very rare in infancy and childhood, and are mostly confined to the myxomatous polypi of the rectum.

B. Inflammatory.—The inflammatory diseases of the intestinal tract not otherwise classified comprise *proctitis*, *appendicitis*, and the various forms of *ileo-colitis*, which are usually designated as dysentery. This latter class represents a number of infectious diseases which are general in type, and each may be caused by a special organism, but in the present state of our knowledge they are most conveniently described in connection with diseases of the intestine.

PROCTITIS.

Proctitis is an inflammation of the rectum, and may be acute or chronic. It is usually coincident with inflammation of the colon, but may occur alone as the result of trauma or the extension of inflammatory processes around the anus.

Symptoms.—The symptoms are essentially pain, traces of blood mixed with mucus, and tenesmus.

Diagnosis.—The diagnosis from reflex inflammatory conditions is made by direct inspection.

Treatment.—The treatment is to keep the child in bed, to keep the fæces soft with laxatives, to cleanse the rectum, and, according to the especial lesions, use astringents or soothing injections. When there is pain or tenesmus, suppositories of opium and cocaine are indicated.

APPENDICITIS.

Etiology.—Inflammation of the vermiform appendix in children is most frequent between the ages of five and fifteen years, and is not of uncommon occurrence. It is very rare under two years. Several cases have been reported as early as seven weeks. Boys are more often attacked than girls, and in about the same proportion as in adults. Functional disturbances of digestion, constipation, diarrhœa, and indiscretions in diet are only remotely connected with the etiology of the disease. Foreign bodies are infrequent, despite the general opinion, but the presence in the appendix of fæcal concretions, often resembling seeds and stones of fruits, are very common, but their exact relation to the primary cause of the inflammation is not clear. Direct trauma, as from a blow in the abdomen, is sometimes a definite exciting cause. The prevailing opinion is to consider the process an infective inflammation, though as yet no specific organism has been shown to cause it. On the contrary, cultures of the colon bacillus alone, or mixed infections of the colon bacillus associated with the streptococcus, pneumococcus, staphylococcus albus or aureus, the bacillus aërogenes, and certain undetermined anaërobic organisms are found. The transformation of the appendix into a closed tube is to be considered as a consequence rather than a cause of appendicitis (Brun).

Pathology.—The pathological conditions occurring in the course of an appendicitis are exceedingly varied, and will only be outlined. In the early stages of a purely appendicular lesion, the appendix is in normal position and may show only slight swelling and congestion. Usually it is voluminous from extensive infiltration of its walls, very turgescent, and often shows spots of ecchymoses with intense congestion of the peritoneal coat. In more advanced stages the mucosa is soft and granular, sometimes gelatinous, with ecchymoses and ulcerations. The muscular coats are thickened and their fibres often separated by minute or extensive accumulations of pus. All these changes may develop in from twenty-four to thirty-six hours after the onset of symptoms. Perforation and gangrene of the appendix, in part or in whole, are simply further steps in the inflammatory process, and, according to Brun, are especially likely to occur in children.

The inflammation is rarely confined to the appendix, but by extension involves the peritoneal coat of the appendix and the peritoneum itself. The subsequent course is very variable. The process may remain local, and lead to a peri-appendicular inflammation, in which the appendix, cæcum, and end of the ileum are shut off from the rest of the peritoneal cavity by false membranes, with or without the formation of pus; or the infection may spread directly from the appendix to the general peritoneal cavity, with the production of general septic peritonitis. The location of the pus in cases of abscess formation depends to some extent upon the location of the appendix, in which there is considerable variation. It may be either iliac, pre-rectal, sub-umbilical, retro-cæcal, or lumbar in its disposition.

A most malignant form of general peritonitis may follow a simple parietal appendicitis without perforation or gangrene of the appendix, and give rise to a clinical picture more suggestive of septicæmia than of peritonitis.

A simple appendicitis, sometimes called *catarrhal*, may produce so much thickening of the walls and infiltration of the submucosa with leucocytes, and subsequent formation of granulation tissue, as to cause an obliteration of the lumen of the appendix. If the lumen is completely obliterated, it may render the patient immune to subsequent attacks, but if only partial, it favors the collection of pockets of pus and cyst-formation, and the organs is a constant source of danger.

Ulcerative appendicitis may be caused by the presence of concretions or by the action of micro-organisms. The presence of concretions, however, may not be accompanied by any inflammatory or necrotic reaction. Typhoid and tubercular ulcerations of the appendix are not uncommon in their respective diseases, and an actinomycotic ulcer has also been described.

SYMPTOMS.—Sudden pain in the abdomen, referred to the umbilicus or to the right iliac fossa, with tenderness on deep pressure in the region of the appendix, associated with a fever of moderate or high grade and gastro-intestinal symptoms, usually mark the onset of an acute inflammation of the appendix. Clinically it is convenient to distinguish between certain types of the disease.

Simple Appendicitis.—This is the least severe of the acute forms, and is characterized by its sudden onset and short duration. Without any prodromal symptoms the child is seized with sudden, severe abdominal pain at about the level of the umbilicus and more to the right than to the left. Vomiting, in some cases only nausea, follows. There is moderate fever. The pain is continuous, and the bowels are usually constipated.

Tenderness over the region of the appendix with a unilateral rigidity of the muscles is the most important sign by which we exclude other causes of abdominal pain. The temperature is moderate, 23.5° to 24.1° C. (100° to 101° F.), lasting a day or two. The acute symptoms subside in from twenty-four to forty-eight hours, but the tenderness is apt to remain for several days, at the end of which time the abdomen is sometimes so relaxed as to enable one by careful examination to feel the slightly thickened appendix. These mild cases probably occur much oftener in children than are diagnosed.

Appendicitis with Periappendicular Peritonitis.—This is the more common form, formerly recognized as *perityphlitis*, and in its mildest grade is not to be distinguished clinically from the severer types of a simple appendicitis. The initial symptoms are more exaggerated, but are the same in character as in a simple appendicitis. The digestive symptoms are much more pronounced. The vomiting is apt to be repeated, and the temperature much higher, 39° to 40° C. (102.2° to 104° F.). The pulse is rapid, 112 to 120 per minute, and the pain and localized tenderness is more intense. After a lapse of some hours, if the abdomen is not too tense, we may sometimes feel in the right iliac region an indistinct thickening, sometimes a definite mass, but the swelling may not be located until the child is under the influence of an anæsthetic. There is apt to be leucocytosis, though one must not be misled by a normal white blood count. In some cases, under expectant treatment; these symptoms will gradually subside in the course of a week or ten days, and with the alleviation of the acute pain and tenderness the swelling of the appendix and surrounding tissue is more readily recognized. In time this mass may entirely disappear so far as external palpation can determine, or, on the other hand, some induration may remain after complete establishment of health.

If the inflammatory process ends in abscess formation the persistence of the fever and the presence of leucocytes aid in the diagnosis. The return of the fever after its subsidence is also indicative of the advent of suppuration. The absence of fever, however, cannot be taken as positive evidence of the absence of pus. If the swelling increases in size or if it remains stationary we strongly suspect that there is a local accumulation of pus. If the pus has once formed it may terminate by absorption or by perforation externally or into some portion of the intestines, vagina, or bladder, but—and this should always be borne in mind by the physician who takes the responsibility of expectant treatment—it may rupture into the peritoneal cavity, usually with the result of a fatal septic peritonitis.

Appendicitis with General Suppurative Peritonitis.—Aside from those cases of general peritonitis which are caused by a rupture of an appendix abscess into the peritoneal cavity, there are others of general peritonitis in which the infection starts from a diseased appendix with symptoms of such rapid development as to indicate a general peritonitis almost from the very beginning. There is sudden severe pain in the region of the umbilicus rapidly spreading over the whole abdomen, but with its greatest intensity on the right side. The vomiting is incessant, and the constipation so sudden and complete as to suggest intestinal obstruction. The temperature rises suddenly to 39° to 40° C. (102.2° to 104° F.), and the pulse is increased to 130 to 140 per minute. The abdomen rapidly becomes rigid throughout, and deep palpation is impossible. The tenderness is general, but most marked on the right side in the neighborhood of the appendix. The symptoms progress rapidly. The vomiting of bile is followed by that of fæces. The abdomen becomes distended. The respiration is labored. Delirium often sets in. Aside from the localized tenderness and rigidity, the examination of the abdomen is negative, for the amount of intraperitoneal fluid is too small to be recognized by percussion, though at times a rectal examination gives evidence of fluid in the cul-de-sacs. Without an early operation, death usually ensues in these cases in from two to four or five days.

There is still another form of appendicitis with diffuse peritonitis, described by Brun, Jalaguier, and others, which is the most malignant and rapid of all, and in its course suggests a septicæmia having its origin in a virulently infected appendix. Its onset is quite different from the type of disease just described. It begins like an attack of acute intestinal indigestion with vomiting and fœtid diarrhœa, rarely, if ever, with constipation. Neither the pain nor the tenderness in the iliac fossa is so marked as in the other cases. The abdomen is soft, flat, and easily palpable, and with almost complete absence of resonance on percussion. The progress is rapid. The features are anxious, leaden-colored, the eyes sunken and surrounded by dark rings. The thirst is keen, the respiration rapid and shallow. There is extreme restlessness. The temperature at the first may reach 39° C. (102.2° F.), but on the second day is more apt to be 37° C. (98.6° F.) or below, in striking contrast to the weak, rapid pulse of 150 to 160. The mind remains clear to the end, without any change in the abdominal symptoms, and death often supervenes within thirty-six to forty-eight hours, rarely extending beyond three or four days.

Chronic Appendicitis.—An appendix once inflamed is prone to recurrent attacks, with the train of events connected with the primary attack. The cause for this probably lies in an obliterative appendicitis with or without adhesions, or perhaps from a localized abscess circumscribed by dense fibroid tissue. The attacks may recur at intervals of a few months, and finally cease or develop into an acute attack which may run a typical course. Chronic appendicitis may, however, manifest itself from the beginning as indefinite twinges of pain in the right iliac fossa, brought on especially by over-exertion and fatigue, without ever developing alarming symptoms, and with but little else in the way of symptoms; on the other hand, these inconstant pains may be prodromal symptoms of a susceptible appendix, which later becomes acutely inflamed.

Diagnosis.—The diagnosis of appendicitis in young children is often made difficult by the inability of the child to locate accurately his sense of pain, and to appreciate the distinction between pain and tenderness. Moreover, the contraction of his abdominal muscles and his inability to relax them at will renders the result of palpation less certain. The presence of fever and localized tenderness in the right iliac fossa are the principal points in the exclusion of hepatic and renal colic. Intestinal colic is more often associated with diarrhœa than with constipation, and here again the localized tenderness is a crucial point in the diagnosis. It is well established that in pneumonia in children the pain is frequently referred to the abdomen, but careful attention to the symptoms and course of the disease will enable one to distinguish between the two conditions. An appendix abscess arising from an appendix unusually placed might easily be confounded with a perinephritic abscess, but in either case operation is indicated, and the error in diagnosis should not prejudice the interests of the child. Appendicitis with the sudden onset of septic peritonitis may readily suggest an intestinal obstruction, but the fever, localized tenderness, and leucocytosis are not likely to occur in the latter condition. In intussusception the straining, tenesmus, and small and frequent discharges of blood and mucus, and the sausage-like tumor, which can often be felt in the region of the transverse colon, usually suffice for a diagnosis. Moreover, appendicitis is very rare under two years, and intussusception is very common. In certain cases the two conditions simulate each other very closely. Perforation of gastric ulcer, pus tubes, and pelvic peritonitis, abscess of the broad ligament, acute pancreatitis, appendicular hypochondriasis and hysteria, which may in the adult be mistaken for appendicitis, are not likely to occur in infants and young children, but in older children should be borne in mind. An acute inflammation of the gall-bladder and appendicitis are often confounded, and an unusual position of the appendix should always be remembered.

Prognosis.—The prognosis depends largely upon how early the diagnosis is made and the skill of the surgeon who is called for consultation and operation. Cases undoubtedly recover in many instances without operation, but the extreme gravity of the results of an extension of the inflammation to the peritoneum, and the realization that this accident may occur at almost any time, even in the cases which are apparently running a mild course, render general statistics of little value in the consideration of an individual case. We should not lose sight of the fact that in infancy we see most frequently the very severe and acute forms of the disease, and there is the special tendency to perforation and gangrene and extension to the peritoneum, with its very serious consequences.

Treatment.—Personally, I consider the disease one for surgical observation and treatment. It is safer to call the surgeon in consultation early than late, and if the diagnosis is determined it is wiser to trust to his judgment as to the expediency of immediate or delayed operation than for the physician himself to attempt to decide. There is no medical treatment other than that which is purely symptomatic. Absolute rest in bed, irrespective of the severity of the symptoms, is of first importance; local applications of ice should be used to control the pain, or, if the child objects, as is frequently the case, hot fomentations should be tried. Cathartics and laxatives should be avoided, and the bowels moved by enemata. Blisters should not be used. Opium is strongly objected to by surgeons as tending to mask the symptoms; if it can be avoided, it is far better to do so; but very small doses, sufficient to keep the patient from excessive pain and fairly quiet, are probably in children a lesser danger than that which would be caused by hours of pain, crying, and intense restlessness. If no operation could be performed I should prefer to trust to opium in the early stages rather than to cathartics.

ILEO-COLITIS.

General Etiology.—Under the term *ileo-colitis* are included all the more marked and grave lesions of the intestine. They are commonly grouped under the name *dysentery*, but as our knowledge of them increases it is becoming evident that any such general term as dysentery is inadequate to cover what will, in all probability, be proved to be a number of diseases, each arising from its own specific organism.

All these forms of ileo-colitis (dysentery) are considered to be infectious. They may occur as acute primary diseases, but are usually secondary to the fermental diarrhœas, and sometimes to the infectious diseases, especially measles. The sporadic cases have not as yet been sufficiently studied bacteriologically to allow us to come to any definite conclusions concerning them etiologically. Flexner has, however, shown that it is entirely possible that two specific organisms may be responsible for both the epidemic and endemic varieties of what has been called tropical dysentery, and that they consist of (1) a *bacillary* and (2) an *amœbic* form.

Amœbic ileo-colitis is so well recognized as a specific infectious disease that it has already been described with the other infectious diseases, on page 501. Our knowledge of the bacillary form is still too limited for us to more than mention it.

The etiology of the other supposed forms of ileo-colitis (dysentery) is so little known, and their lesions are so varied, that it would be impossible to classify them in detail. Practically, also, we can divide them only in such a general way that for the present they had better be described as in the case of the fermental diarrhœas, under diseases of the gastro-enteric tract.

Variations in Type.—The divisions which have been adopted to simplify the subject are (1) *simple catarrhal inflammation*, which includes the non-ulcerative form of follicular inflammation, (2) *follicular inflammation*, and (3) a *pseudo-membranous inflammation*.

Although these three forms differ essentially in their prognosis, they are so often represented by the same symptoms that they can be differentiated only in the most general way. A symptom common to all these diseases is found in the temperature, which, although not necessarily high, is, as a rule, raised throughout the whole course of the disease. In this way we can usually differentiate these diseases from the non-inflammatory conditions.

The pseudo-membranous form of ileo-colitis may be primary or secondary. In the primary form it represents what is usually spoken of as epidemic or sporadic dysentery. The secondary form is that which follows certain infectious diseases.

The pathological lesions found in connection with the catarrhal and non-ulcerative follicular and the ulcerative follicular inflammations approach each other so closely and the symptoms are so similar that the clinical distinction between the two conditions is very difficult.

PATHOLOGY.—In the pseudo-membranous forms of ileo-colitis, the ileum and colon are chiefly affected. The disease is characterized by the presence of a membrane on the surface of the mucous membrane, which extends into it, and is due to a combination of fibrous exudation and necrosis. The pathological conditions found in the various forms of ileo-colitis may best be studied in connection with the histories, autopsies, and figures of the cases presently to be reported.

Acute Ileo-Colitis.—SYMPTOMS.—The symptoms of the various forms of acute inflammatory ileo-colitis vary greatly, as a rule, but in a general way they can be recognized by a group of symptoms which differ from those of the non-inflammatory diarrhœas, spoken of as fermental diarrhœa and cholera infantum. The symptoms, however, of these different forms of ileo-colitis are very unsatisfactory and unreliable for differential diagnosis.

The onset of the disease may be preceded by a fermental diarrhœa, or it may be acute from the beginning and have prodromal symptoms of no more than a few hours. The temperature is elevated, the pulse is accelerated, and the infant loses rapidly in weight and strength. The discharges are perhaps ten or twenty, or even more, in the twenty-four hours, but are comparatively small in amount. Where the lesions are in the rectum there is tenesmus both before and after the discharge, and in the beginning of the attack an almost constant desire to have a movement. The discharges contain fæcal matter at first, but soon become small, and consist of mucus, sometimes with pus, blood, and shreds of membrane. The odor may be very offensive, but when the mucus predominates there is very little odor. The color and consistency are extremely variable, but generally the consistency is lessened and the color is a mixture of green, brown, and yellow. The blood is usually from congestion of the blood-vessels and straining, rather than from ulceration, so that we cannot determine from the presence of blood whether ulceration is present or not. At first the abdomen may be soft and not tender, but later in the disease it becomes distended, tympanitic, and somewhat tender, especially along the course of the colon. Vomiting may occur at times. In severe cases the child is very restless, and there may be delirium and convulsions. The appetite is usually much lessened. The urine is nearly always diminished in quantity, is high-colored, and sometimes contains a small amount of albumin, especially when the temperature is high. Casts may also be found. The renal condition in these cases is a degenerative one, and not a true nephritis. Acute nephritis is rare. When there is much tenesmus and straining, and when the discharges are especially frequent, prolapse of the rectum may occur. The discharges often cause great irritation around the anus and on the buttocks.

Although there are no symptoms typical of the different forms of acute ileo-colitis, yet their clinical pictures differ somewhat.

It is usually found in the simple catarrhal ileo-colitis, when ulceration has not taken place, that the symptoms are milder and that there is apt to be vomiting. The cases generally begin to improve in one or two weeks, and recover entirely in another week. An intestinal disturbance of a mild character may result, however, and prolong the disease. The children are usually a long time in regaining their strength, and relapses are quite common in this form if the diet is not carefully regulated. Sometimes, however, simple catarrhal ileo-colitis may be represented by symptoms of a very severe type, and it may run a rapid course, and end fatally.

When follicular ulceration has taken place the stomach is not apt to be much involved, the temperature is not, as a rule, high, and the course of the disease is rather slow, irregular, and prolonged. The infant fails steadily and commonly dies. A remission in the symptoms and an improvement in the character of the fæcal discharges should lead us to infer that ulceration has not taken place. When the inflammation is simply follicular, without ulceration, the cases are very apt to recover.

Pseudo-membranous ileo-colitis is rare in infants, but when it occurs it is the most severe of all the forms. I have already stated that it is this form which is usually spoken of as epidemic or sporadic dysentery. The temperature is high,—39.4°, 40°, or 40.5° C. (103°, 104°, or 105° F.). There are apt to be blood and membranous detritus in the discharges. The progress of the disease is usually rapid and without remission, and death may take place in a week or ten days. The nervous symptoms, such as restlessness and delirium, are quite prominent.

Diagnosis.—These forms of ileo-colitis are diagnosticated from the fermental diarrhœas by the continued heightened temperature, the more frequent discharges, the small amount in each, the presence of blood or membrane, and the tenesmus. They may be differentiated from cholera infantum by the absence of continuous and excessive vomiting and by the serous discharges of the latter disease. The diagnosis, however, can be made positively only by finding shreds of membrane in the discharges.

PROGNOSIS.—The prognosis of ileo-colitis, when ulceration has not occurred, is usually favorable, the duration of the disease being a few weeks. Some cases, however, are more severe, and sometimes prove fatal in a few days. When there is ulceration, the prognosis is rather unfavorable. When there is a diminution in the frequency of the discharges and fæcal matter begins to reappear, and when the nervous symptoms and exhaustion lessen, the prognosis is good; but when the symptoms increase in severity and the face looks pinched, when intractable vomiting arises and the nervous symptoms predominate, the prognosis is very unfavorable.

The prognosis is less favorable when the ileo-colitis is complicated by broncho-pneumonia or tuberculosis. It is much influenced by the time of the year at which the attack takes place, the prognosis being worse if the disease occurs at a time when the convalescence is during a long heated period. The prognosis is also worse when the infants have to be treated in crowded cities and in the midst of unsanitary surroundings.

TREATMENT.—The treatment of these forms of ileo-colitis should usually be in the beginning the same as has already been described for fermental diarrhœa. It may in this sense be spoken of as prophylactic, for in a large number of cases the organisms which produce ileo-colitis find a means of entrance through the irritated mucous membrane produced by a preceding fermental diarrhœa. When the case is seen in its earlier stages, a mild laxative should be given, in order to clear away, so far as possible, the pathogenic organisms, which are present in large numbers. Small doses of castor oil act most efficiently, and can usually be given, especially to infants, without causing nausea or gastric irritation.

In addition to this treatment by the mouth, thorough irrigation of the colon should be employed. This should be done twice in the twenty-four hours with warm sterilized water containing 3.75 grammes (1 drachm) of borate of sodium to the pint of water. One or two gallons of water should be allowed to flow in and out of the intestine at each irrigation. After the irrigation, small enemata of thin mucilage, about 120 c.c. (4 ounces), containing 15 grammes (½ ounce) of bismuth in suspension, may be given once in three or four hours.

According to the degree of pain, restlessness, and general discomfort, a slight amount of opium can be given in these injections, but in all cases this drug should be administered with great care; 0.03 c.c. (½ minim) of tincture of opium in the first year, and 0.06 c.c. (1 minim) in the second year, once in five or six hours, will usually be sufficient to make the infant comfortable. The effect of the opium should be carefully watched, and the dose increased or decreased as is necessary.

When the tenesmus is extreme, it is well to use suppositories containing from 0.015 to 0.03 gramme (¼ to ½ grain) of cocaine. These suppositories will often give great relief if the painful lesions are mostly in the rectum, but when the lesions are higher in the colon they are not of much value, and when the tenesmus is continuous and exhausting, and the suppositories ineffective, a subcutaneous injection of morphia, 0.003 gramme ($\frac{1}{20}$ grain), with atropia, 0.0003 gramme ($\frac{1}{200}$ grain), can be given.

The use of antiseptics by the mouth I do not recommend. Bismuth can be given by the mouth with some advantage in these cases, but the dose must be considerable to accomplish good results. 1.87 gramme ($\frac{1}{2}$ drachm) in the twenty-four hours should be given to a child a year old, and for older children the dose should be proportionately increased. Alcoholic stimulants can be given with benefit at all stages of the disease if there is evidence of a weakened heart, or if much exhaustion is present.

A very limited amount of food of any kind should be given during the first twenty-four hours. Sterilized water containing an alcoholic stimulant and barley-water had better be given at first, as it has been found that when a sterile liquid is taken by the mouth the number of bacteria in the intestine diminishes rapidly. After this preliminary treatment if a perfectly fresh milk can be obtained it may be used, if sterilized and modified in its various elements so as to be adapted to the digestion of the especial case. A moderate percentage of fat and sugar, such as 3 and 5, and a proteid percentage of about 2, is a very good prescription to begin with. Weak broths can also be given.

Chronic Ileo-Colitis (*chronic dysentery*).—ETIOLOGY.—In some cases of ileo-colitis, after the acute symptoms have ceased, the diarrhœa continues for many months, and the disease becomes chronic.

PATHOLOGY.—The pathological conditions most commonly found in these chronic forms of ileo-colitis are great thickening of the muscular tissue, pigmentation of the mucous membrane, and very extensive ulceration.

SYMPTOMS.—There is no especial pain or tenderness, and the temperature may be normal. The appetite often returns, but the child does not gain in weight, or it loses. The discharges are not so frequent as during the acute stage of the disease, varying from six or eight to two or three in the twenty-four hours. The discharges have a lessened consistency, and contain mucus and undigested food. There may at times be exacerbations of the symptoms, and the children are very apt to die of some intercurrent disease. The duration may be many months.

TREATMENT.—The treatment is, if possible, a change of air, and is otherwise essentially diatetic.

The rules already given for the treatment of the chronic forms of fermental diarrhœas are also applicable to this class of cases.

As illustrations of the difficulty and in many instances the impossibility of diagnosticating intestinal lesions, the following cases, which were under my care, are of interest.

One of these cases was that of a little girl, five years old, who during the hot weather in August had been having a slight attack of fermental diarrhœa, which began with vomiting, headache, and a slight rise of temperature lasting a few hours. This was soon followed by four or five greenish-yellow discharges in the twenty-four hours, and a normal temperature. The diarrhœa diminished in two or three days, and the child seemed much better, but after a few days she was suddenly attacked with a temperature of 39.4° to 40° C. (103° to 104° F.) and with frequent discharges of mucus and blood. She lost rapidly in weight, and looked very sick. After twenty-four hours, however, the movements became normal; and on the following day, although left weak and prostrated, she seemed perfectly well, and had no return of the attack. During the acute symptoms it seemed as if she were attacked by one of the more severe forms of colitis, but the rapid recovery left the diagnosis very doubtful.

The next case was that of a child, seven years old, who entered my wards at the Boston City Hospital with a history of having had a slight diarrhœa for a few days. The temperature was but slightly raised. The movements were infrequent, of a greenish-yellow color, and contained no blood or membrane, and scarcely any mucus. The child seemed fairly well on entering the hospital, but during the following few days became much exhausted. Although no other intestinal symptoms appeared, he sank rapidly, and died apparently from exhaustion. The autopsy showed extensive lesions of the whole colon, the mucous membrane was greatly thickened, and there were numerous ulcerations.

The third case was that of a boy, four years old, who was brought to the Children's Hospital for frequent vomiting following an attack of diphtheria. During the first three weeks that he was in the hospital the vomiting was the chief symptom. He was fed by nutritive enemata, and improved in his general strength. Later, however, he became very much emaciated, the vomiting increased in frequency, and a few days before he died there was a slight diarrhœa. The temperature was normal or subnormal during the whole course of the disease. During the last four or five days the symptoms had pointed almost entirely to the stomach, but the post-mortem examination showed nothing abnormal in the stomach, lungs, heart, kidneys, or spleen. The mesenteric glands were swollen in the region of the ileo-cæcal valve. The walls of the ileum and colon were thickened and reddened. There was a slight deposit of fibrin over part of the mucous membrane of the ileum. The lower 35 cm. (13¾ inches) of the colon were found to be much thickened, the inner surface was of a dark-greenish color, and beneath it the tissue was deeply injected. The thickening seemed largely due to an exudation on the mucous membrane, which could not be torn away. The thickening ended quite sharply, but on some of the valvulæ conniventes above a similar membranous deposit could be found. In the colon the thickening was most marked in the cæcum and the rectum, and least so in the transverse colon, and the process seemed older than in the ileum. Cultures from the various organs were negative. Various organisms were found in the ileum, but none that seemed to be of especial significance.

The following cases and figures illustrate varieties of ileo-colitis and show how with our present knowledge it is usually impossible to diagnosticate the especial lesions during life.

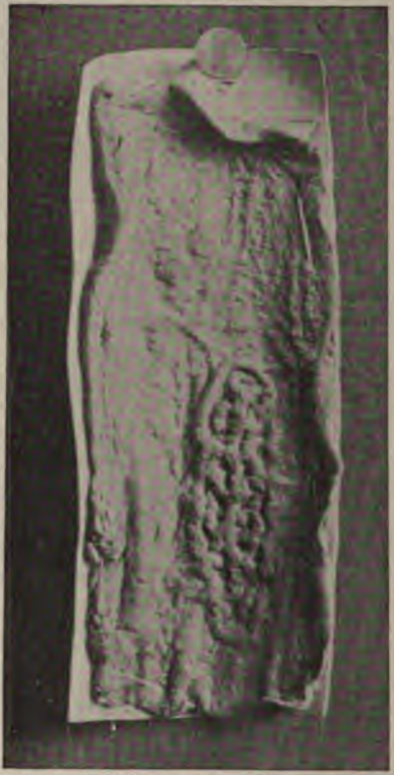
The first specimen (Fig. 165) is a portion of the colon of an infant who during life had only a slight diarrhœa.

The lesion is quite marked and simulates closely the hyperplasia of Peyer's patches which is commonly seen in typhoid fever; but in this case it represents merely intestinal irritation.

This next specimen (Fig. 166) was found at the autopsy of a little girl, three years old, who had been under the care of Dr. Webber.

The child was attacked with excessive vomiting after eating pigs' feet, and the vomiting continued until her death, five days later. The lesions were chiefly in the upper part of the colon, and consisted of a general non-ulcerative follicular inflammation. The hyperplasia of Peyer's patches was extreme.

FIG. 165.



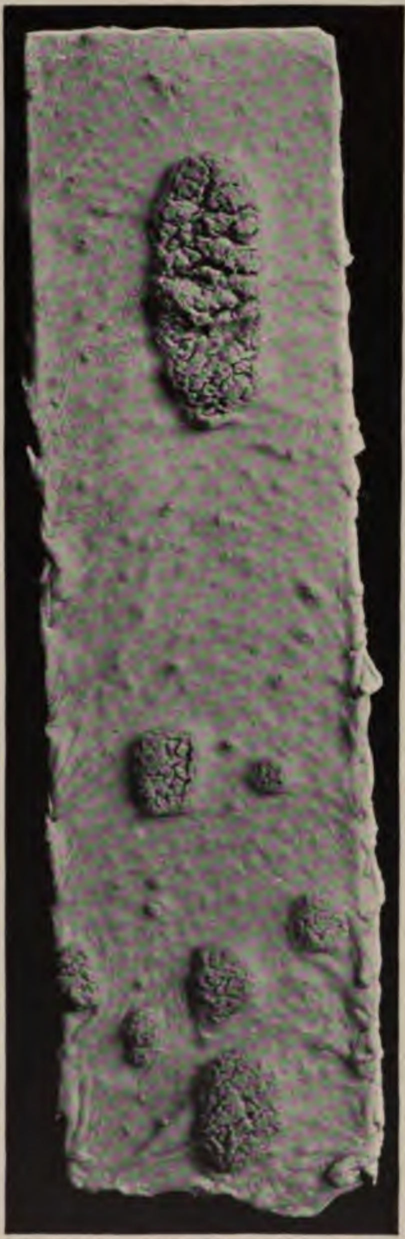
Hyperplasia of the lymph-follicles. Warren Museum, Harvard University.

The following interesting specimens of lesions of colitis occurred in the hospital service of Dr. Northrup, and are now preserved in the Museum of the College of Physicians and Surgeons, New York.

Fig. 167 shows an acute catarrhal follicular inflammation without ulceration.

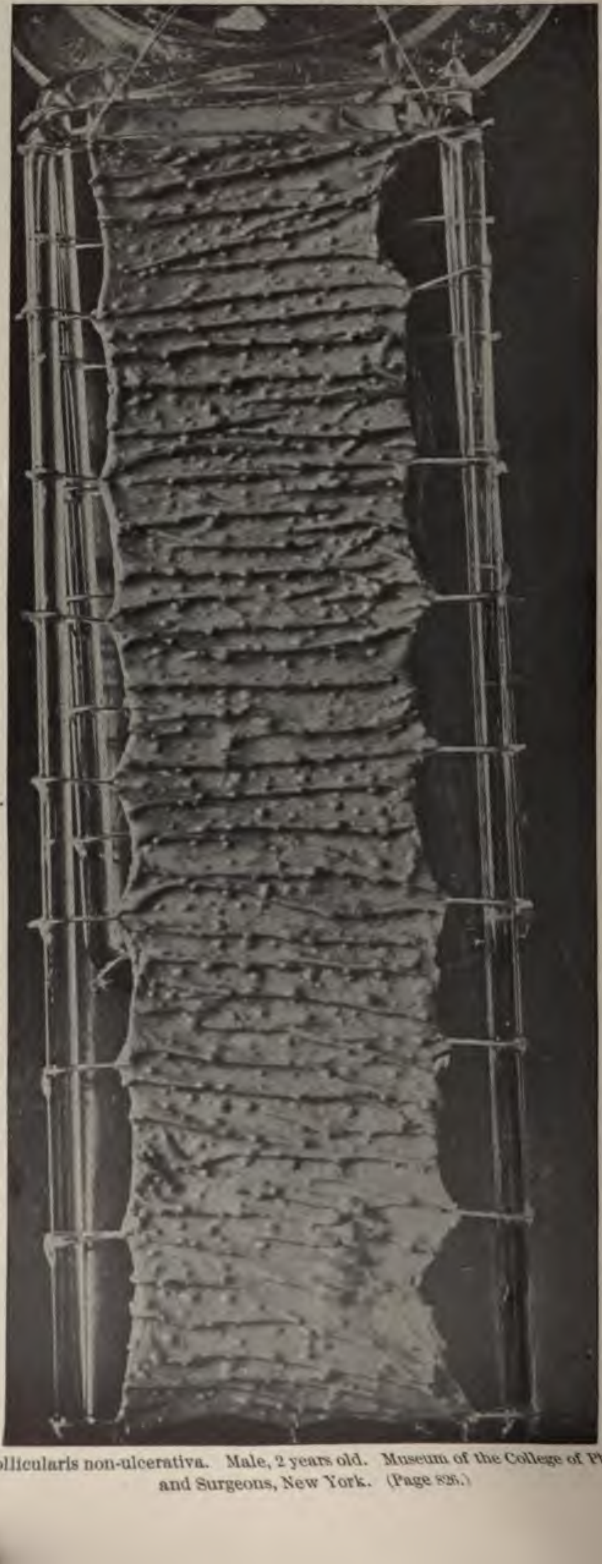
The infant, a male, two years old, had a history of diarrhœa and general debility lasting two weeks. While in the hospital he had a continued high temperature, which at one time reached 40° C. (104° F.). The symptoms were mostly of a cerebral type, and the abdominal symptoms were not severe or prominent enough to indicate the marked lesions which were found at the autopsy. The post-mortem examination showed the following conditions: Brain normal. Stomach congested. The small intestine contained a large amount of thick mucus. The solitary follicles were enlarged, rather more in the upper third of the intestine. Peyer's patches were markedly

Fig. 166.



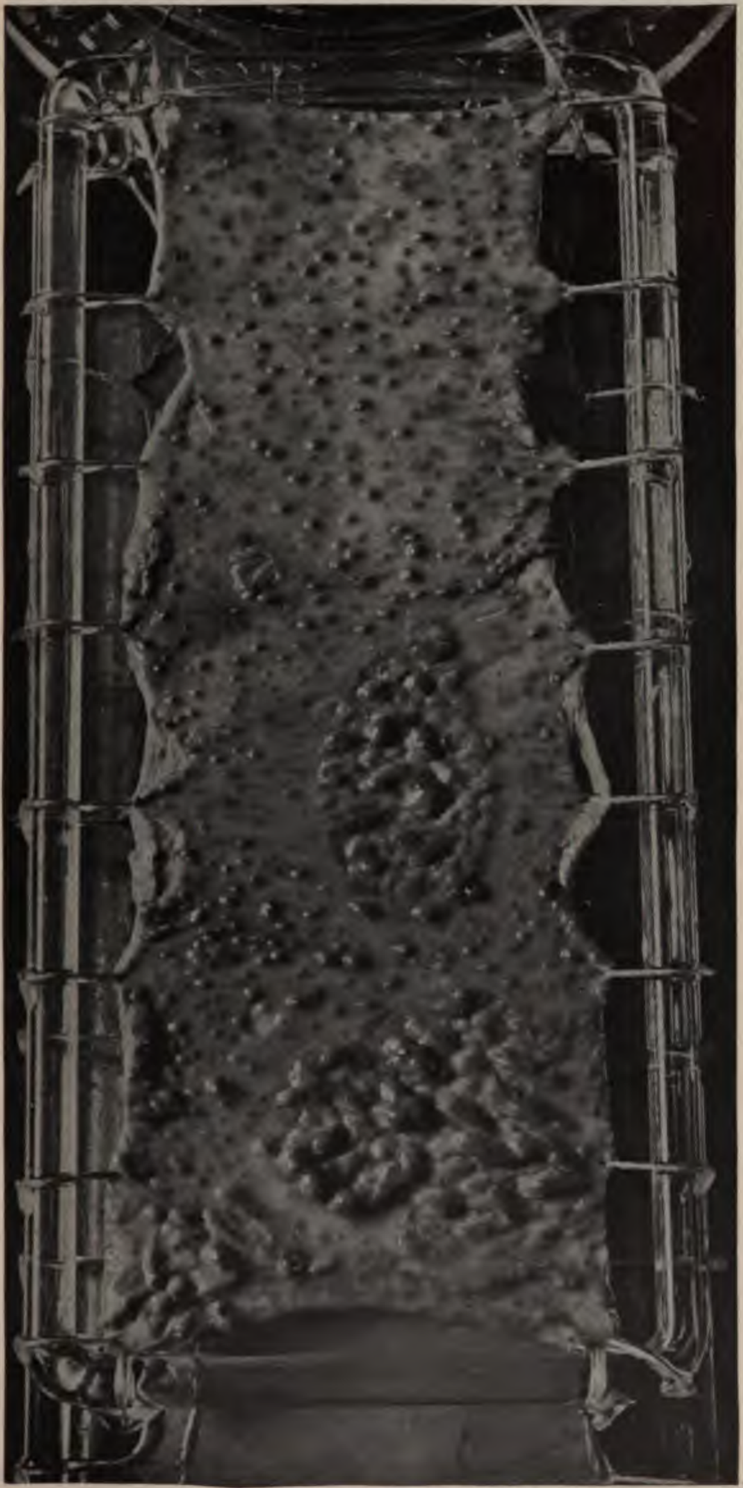
Non-ulcerative follicular inflammation. Simple hyperplasia of lymph-follicles. Female, 3 years old. Warren Museum, Harvard University. (Page 826.)

FIG. 167.



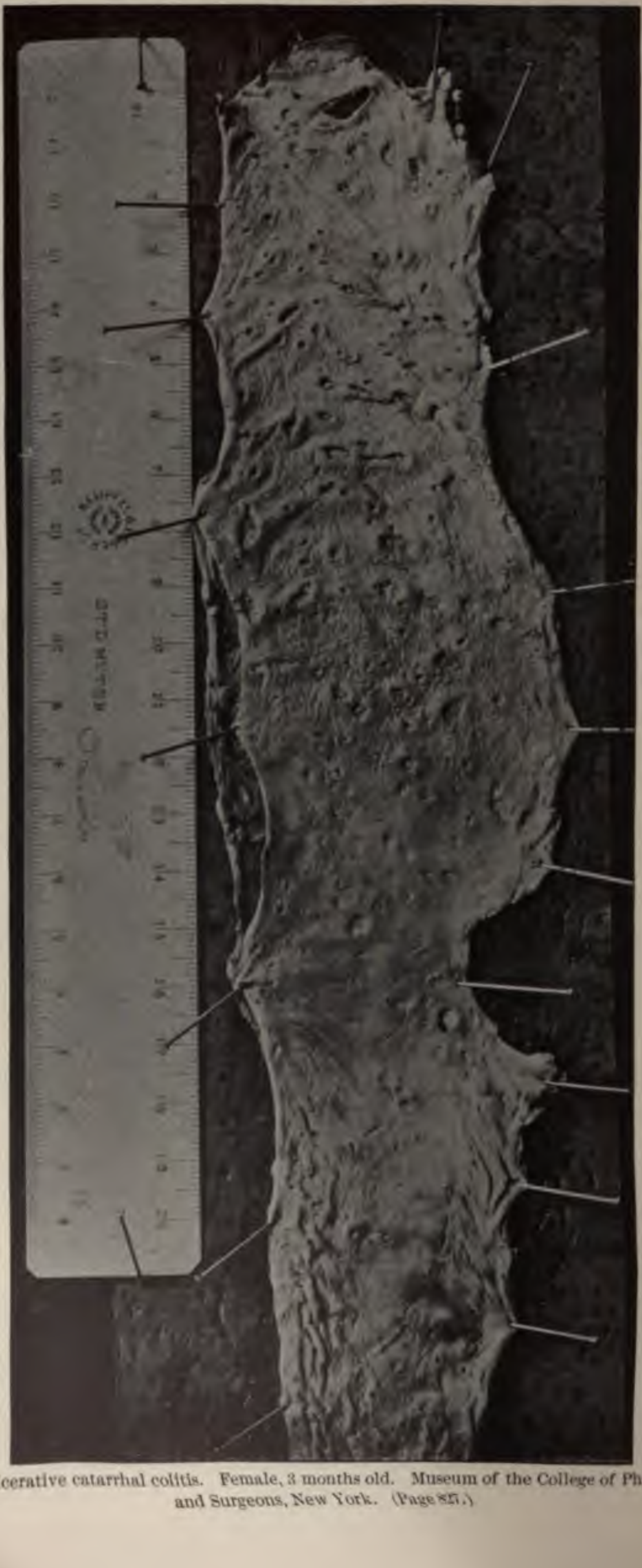
Colitis follicularis non-ulcerativa. Male, 2 years old. Museum of the College of Physicians and Surgeons, New York. (Page 826.)

FIG. 168.



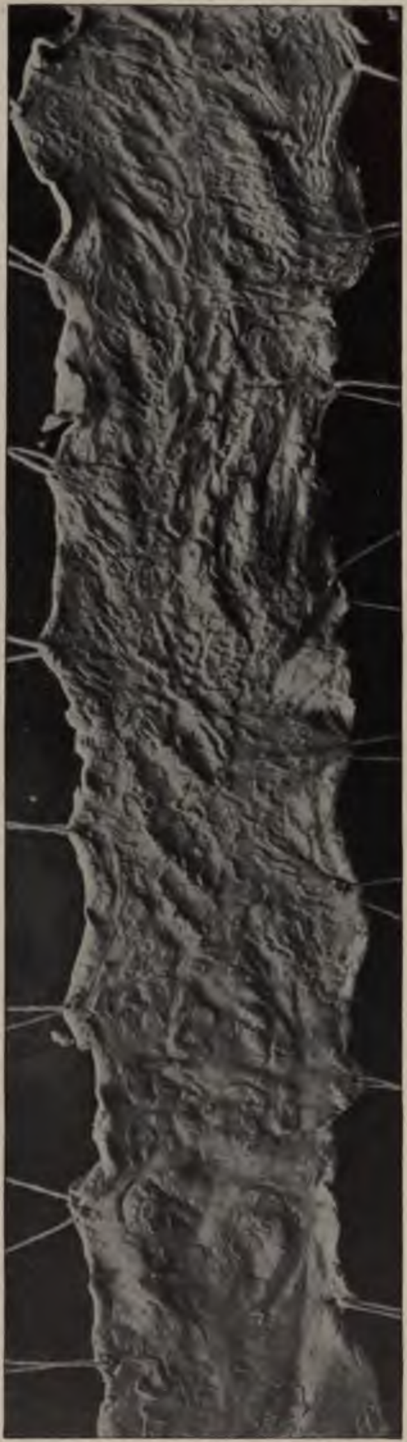
Colitis follicularis non-ulcerativa. (Page 827.)

Fig. 172.



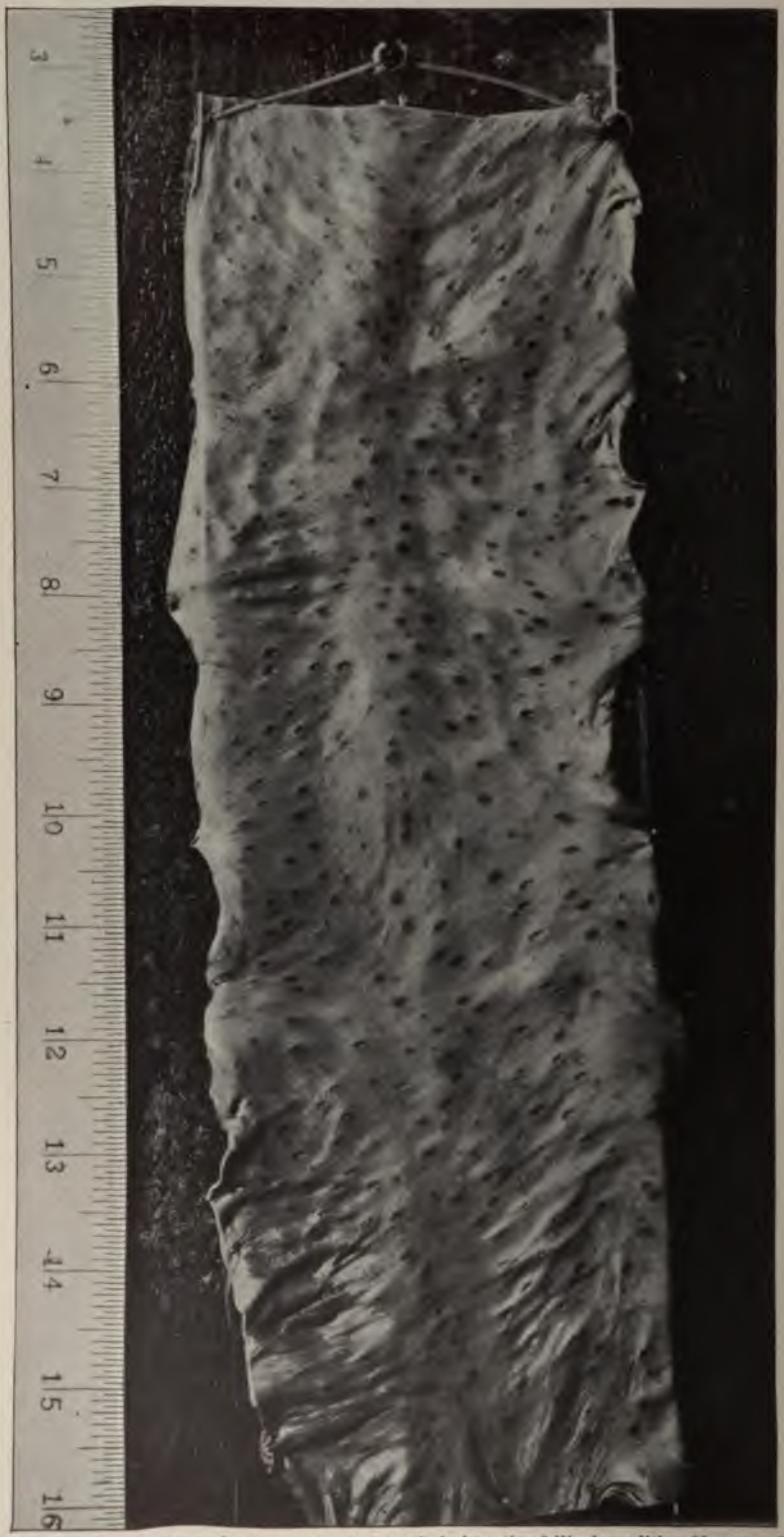
Acute ulcerative catarrhal colitis. Female, 3 months old. Museum of the College of Physicians and Surgeons, New York. (Page 825.)

FIG. 173.



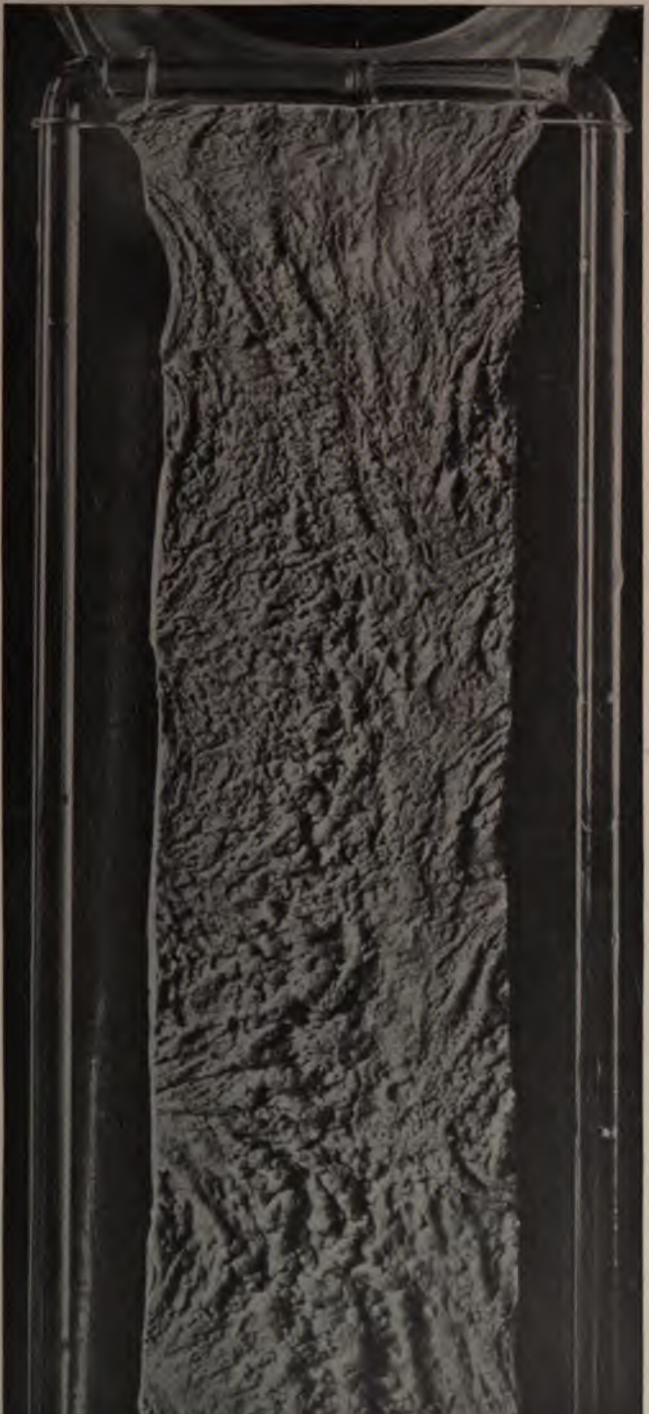
Inflammation of follicles and surrounding parts of colon. The process has gone on to necrosis. Female. 3 months old. Warren Museum, Harvard University. (Page 827.)

FIG. 174.



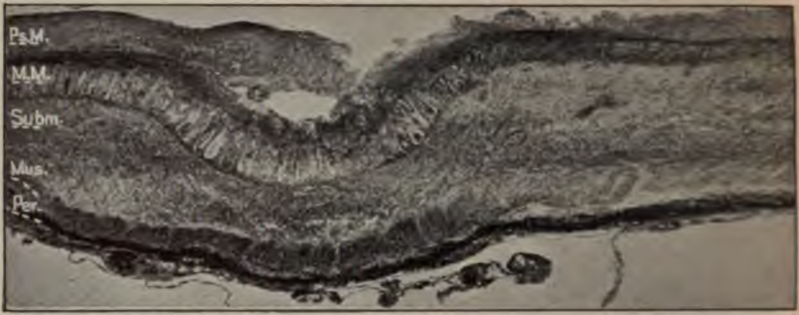
Pigmented follicular ulcers of colon. Chronic catarrhal ulcerative follicular colitis. Museum of the College of Physicians and Surgeons, New York. (Page 828.)

FIG. 175.



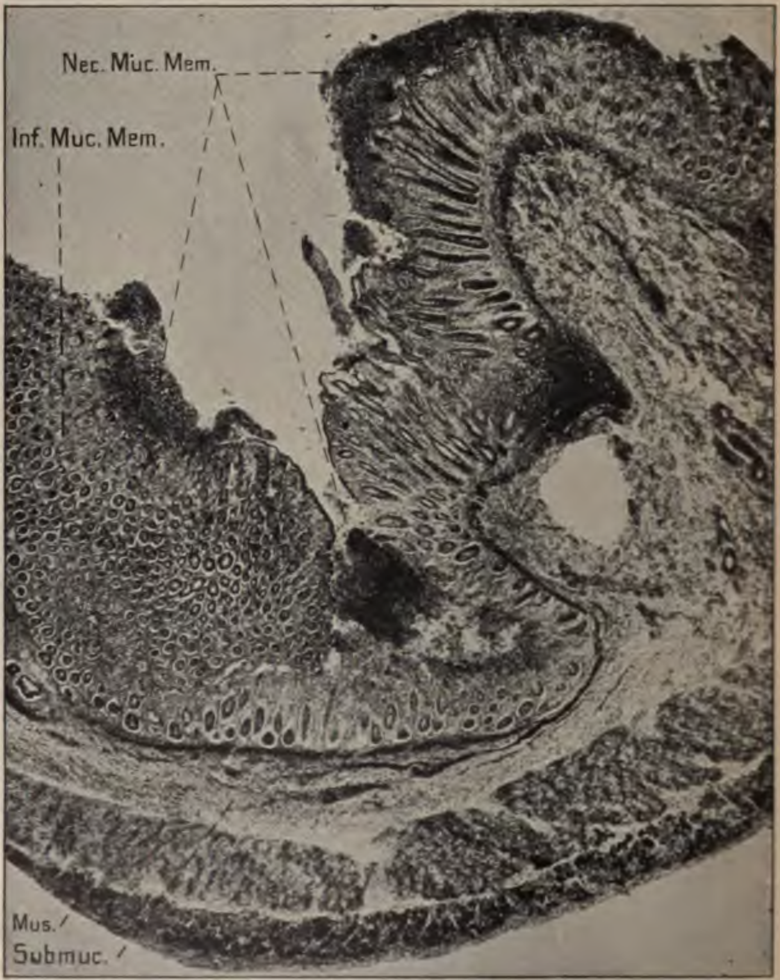
Pseudo-membranous colitis. Child, 3½ years old. Museum of the College of Physicians and Surgeons, New York. (Page 828.)

Fig. 176.



Pseudo-membranous colitis. Female, 4 years old. Ps. M., pseudo-membrane; M. M., mucous membrane; Subm., submucosa; Mus., muscle; Per., peritoneum. (Page 828.)

Fig. 177.



Nec. Muc. Mem., necrotic mucous membrane; Inf. Muc. Mem., inflamed mucous membrane; Mus., muscle; Submuc., submucosa. (Page 828.)

swollen, and a few solitary follicles appeared to be ulcerated. The mesenteric lymph-glands were enlarged. The mucous membrane of the colon was swollen ; the follicles were enlarged and somewhat pigmented, but not ulcerated.

Fig. 168 shows another portion of the colon taken from the same infant.

The solitary follicles are very much enlarged, and in Peyer's patches, which are in the middle of the specimen, the hyperplasia is of a very high degree.

Figs. 169 and 170 illustrate microscopic sections of this form of follicular inflammation. The former showed the great enlargement of the lymph-follicles; the latter showed the inflamed condition of the mucous membrane as well as the enlarged lymph-follicles.

The next specimen (Fig. 171) was taken from an infant sixteen months old.

The infant before entering the hospital had had occasional attacks of diarrhœa for three months, presumably caused by improper feeding. Soon after entering the hospital it rapidly grew worse and died.

The autopsy, made by Northrup, gave the following results : No tubercular lesions. Bronchial lymph-follicles enlarged. Small intestine showed much swelling and congestion of Peyer's patches, but no ulceration. The colon showed extensive follicular ulcerations. In the small intestine and the colon were found masses of strings of greenish mucus ; no blood.

The next specimen (Fig. 172) was taken from a female infant, three months old.

The infant on entering the hospital was somewhat rhachitic, emaciated, and fretful. There were no vomiting and no fever. It took very little nourishment, and at this time was having one large, watery, fæcal discharge daily. The fæcal movements were greenish yellow. The infant apparently improved for about a week. The temperature was then found to have risen, and during the next week it varied from 36.6° to 37.7° C. (98° to 100° F.). During the next week the temperature was sometimes subnormal. At the end of three weeks the infant began to fail rapidly without any discoverable cause, and died suddenly.

The autopsy was made by Northrup, and showed the following lesions : the mucous membrane of the ileum was swollen, and the lymph-follicles were enlarged, but not ulcerated. The report of the examination of the colon was as follows : numerous ulcers, some round and some irregular in shape ; an increased production of mucus ; a profuse growth of connective tissue between the tubules, with disappearance of the tubules ; necrosis of the new tissue so as to form ulcers ; the solitary follicles swollen, but not concerned in the formation of ulcers, which were simply necrotic. No amœbæ found. The process was one which would ordinarily come under the head of acute catarrhal colitis.

The next specimen (Fig. 173) was taken from an infant three months old, in the hospital under the care of Dr. Holt.

The child had no acute symptoms, but had never been well, and for some time had been losing in weight and strength. It entered the hospital for vomiting and diarrhœa. Nothing was found on physical examination. While in the hospital it had from six to eight loose greenish discharges in the twenty-four hours, and vomited occasionally. Its temperature varied from 37.2° to 38.3° C. (99° to 101° F.). It gradually failed, and died twelve days after entrance.

The post-mortem examination showed extensive follicular ulcerations of the colon, especially in the lower part of the specimen, where there was a large ulcer. The tissues around the follicles are also involved, and the process had gone on to necrosis.

The next specimen (Fig. 174) was taken from a male infant six months old.

The infant entered the hospital in a very wasted condition, and died in a few days without any special abdominal symptoms. The autopsy, made by Northrup, showed numerous superficial abscesses on the body, a general bronchitis, and a beginning broncho-pneumonia. The lesions in the intestine were an inflammation of the solitary follicles of the ileum and of the colon, with small ulcerations at the apices of the follicles in the colon, no ulcers being present in the ileum. In the specimen the ulcers were pigmented, which denoted a chronic condition. The apices of the follicles are sometimes found pigmented as the result of post-mortem changes, and may simulate these ulcerations.

The next specimen (Fig. 175) was one of pseudo-membranous colitis.

This child, three and a half years old, a patient of Northrup's, entered the hospital in a very reduced condition following an attack of whooping-cough. It was attacked with diphtheria, and during the ten days that it was suffering from this disease there was a slight amount of diarrhœa, but no pain and no tenesmus.

The autopsy showed a pseudo-membranous inflammation through the whole length of the colon, most marked in the lower third. The other organs were normal. The microscopic examination of the colon confirmed the diagnosis of pseudo-membranous colitis.

Fig. 176 shows a microscopic section of another case of pseudo-membranous colitis.

A girl, four years old, had always been delicate. She had pneumonia twice in her fourth year. Eight days before her death she was attacked with vomiting and diarrhœa. There was blood in the fæcal discharges. The pulse was rapid. The loss of strength and the pallor were marked. The eyes were sunken, and the tongue was dry. On the last day of her life she became very feeble, and died in convulsions. Early in the disease the discharges were frequent. Later, they were from four to six daily, and were accompanied by tenesmus and tenderness of the abdomen.

The autopsy showed that the mesenteric lymph-follicles were not much enlarged; the follicles in the colon were slightly enlarged. The whole intestine was injected in patches, and contained fæcal masses of a yellowish color. The large intestine was filled with large quantities of fæces of foul odor and colored by bismuth. The whole surface was rough, and did not look like a mucous membrane, but rather as though a thin layer of gelatin had been poured over it. This film could be pulled away with the forceps. The solitary follicles were enlarged. The microscopic section of this specimen showed a marked fibrino-purulent exudation, forming a membrane which characterized the disease as pseudo-membranous colitis.

In connection with the pseudo-membranous condition shown in Fig. 175, another specimen (Fig. 177) is of interest.

This specimen was taken from a child, three and a half years old, who first had whooping-cough and was then attacked with diphtheria. During the course of the disease the temperature was raised continuously, at times being as high as 40° C. (104° F.). During this attack it had diarrhœa with blood in the discharges, but no pain or tenesmus and no other symptoms of colitis.

The autopsy showed a broncho-pneumonia, and a normal condition of the stomach and small intestine. The colon showed an apparent exudation, which simulated that of a pseudo-membranous colitis so closely that before the microscopic examination was made it was supposed to be identical with the pathological lesions found in the case of pseudo-membranous colitis (Fig. 175). The surface appearance in the fresh specimen was identical. Under the microscope, however, the lesion proved to be only a superficial necrosis of the mucosa, with swelling of the lymph-follicles.

This case should impress upon us how important it is not to rely upon the macroscopic appearances of intestinal lesions without the corroboration of a microscopic examination.

ANIMAL PARASITES.

The animal parasites which are found in the intestines of infants and children are the same as those which occur in older patients. The only ones, however, which are common and important enough to speak of are the *oxyuris vermicularis* (pin-worm), the *ascaris lumbricoides* (round-worm), the *tænia solium*, and the *tænia mediocanellata* (tapeworms).

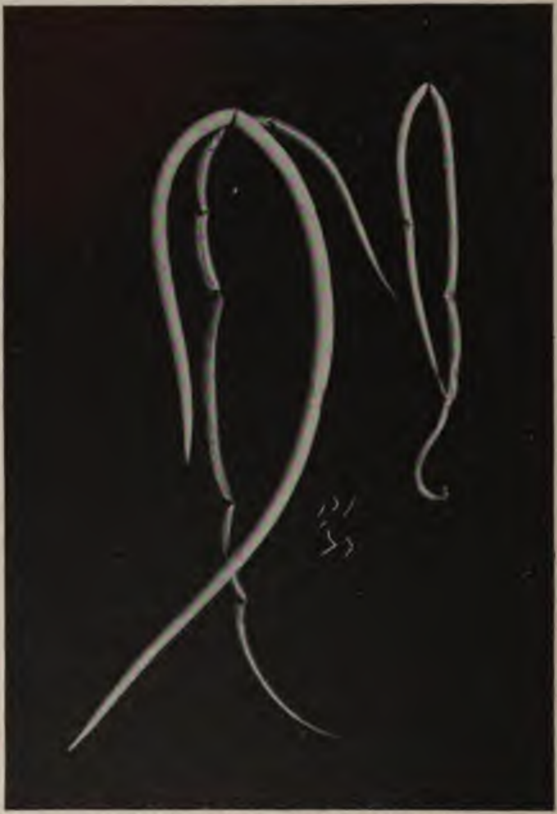
Oxyuris Vermicularis.—The oxyuris vermicularis is a minute worm, which looks like a little piece of white thread. The female is from 0.6 to 1.2 cm. (¼ to ½ inch) in length. The male is about one-third as large, and has the tail rolled into a spiral. Their development takes place in the large intestine, and the mature worms deposit their eggs in the rectum. They enter the intestine through the mouth, and children are very apt to reinfect themselves by carrying the eggs on the fingers or under the nails to their mouths.

These worms sometimes exist in large numbers, and their development is so rapid that it is often difficult to dislodge them completely. The most common symptom of the oxyuris is an intense itching about the anus. The sleep of the child is disturbed by this irritation, and various nervous symptoms develop in children who are infested with this parasite. Thus incontinence of urine sometimes results. In girls the parasite, by migrating from the anus to the vulva, may cause a vulvo-vaginitis.

Diagnosis.—The diagnosis of the presence of these, as of other intestinal parasites, can be made only by finding the worm or its ova in the intestinal discharges. When they are suspected, an enema of clear water should be given. If the parasites are present, they will be dislodged, and careful inspection will disclose their presence. Whenever there are symptoms of reflex irritation in the neighborhood of the anus or the genital organs, the oxyuris should be suspected and sought for. The parasites can often be found in the fæcal discharges, and in some cases they can be seen by simply stretching open the anus and examining the mucous membrane of the rectum.

TREATMENT.—Although most of the worms are in the rectum, yet they also infest the upper parts of the intestine, and therefore cannot be reached by enemata. In many cases enemata of salt-and-water are sufficient to produce a cure, but in some cases the salt, even in small amount, is so irritating that it cannot be used. Infusions of quassia may also be employed as enemata. One of the most effective methods of dislodging the parasite is to give every evening at bedtime an injection of 60 c.c. (2 ounces) of sweet oil. This is allowed to remain in the rectum for five or six minutes, and a large enema of water is then used to wash out the oil, which usually carries with it the parasites from the lower colon and the rectum. Care must be taken in regard to cleanliness, so as to prevent reinfection.

FIG. 178.



Oxyuris vermicularis. Ascaris lumbricoides.

When this treatment is not sufficient, lozenges of santonin, 0.01 to 0.03 gramme ($\frac{1}{6}$ to $\frac{1}{2}$ grain), according to the age, may be given two or three times daily.

Every two or three days a cathartic, such as castor oil or calomel, should be given. Care must be used in giving santonin not to produce symptoms of poisoning, such as gastro-enteric irritation, dizziness, and yellow vision. This occurrence, however, will not be common if in each case the effect of the drug on the child is carefully watched. Serious symptoms, such as convulsions, have been caused by a lack of care in using this drug in young children.

Under this treatment, aided by high rectal injections, the worms can in most instances be eradicated. I have, however, met with very intractable cases in which months and even years had elapsed before treatment of any kind was successful. In such cases temporary relief can be obtained by giving the child each night, or two or three times a week, a small enema of oil.

Ascaris Lumbricoides.—The ascaris lumbricoides is a long, cylindrical, yellowish-white or reddish-yellow worm, pointed at both extremities. The male is distinguished from the female by the fact that it is smaller and is always rolled upon itself, while the female is straight. The length of the male is from 10.4 to 18 cm. (4 to 7 inches), and that of the female from 15.5 to 28.5 cm. (6 to 11 inches). The eggs of this worm are oval in shape, 0.075 mm. long and 0.058 mm. wide. When they are first passed they are almost transparent, but they soon become yellowish and opaque. These eggs are not developed within the intestine, but may pass out with the fæces. They are very tenacious of life, and may develop under favorable circumstances after many years. The embryos are developed outside of the body, and reach the intestine with the drinking-water, where they develop into the mature worm.

The habitat of the worm is usually in the small intestine. It may, however, pass through the rectum either with the fæces or alone, and may migrate into the stomach, œsophagus, or nose. Sudden death has resulted from the entrance of these worms into the air-passages. They may also at times enter the common and cystic bile-ducts, and they have even penetrated farther and caused abscess of the liver. There is no danger of their perforating a normal intestine, but when ulceration has been present perforation has occurred.

SYMPTOMS.—There are no especial symptoms produced by this worm, and we can diagnosticate its presence only by seeing it or by finding the eggs in the fæcal discharges. The worm may in some instances produce a feeling of discomfort or even colic in the region of the umbilicus. Neither of these symptoms, however, can be depended upon, and an anthelmintic is required to determine whether the parasite is present. In certain cases convulsions in children have been followed by the passage of a lumbricoid worm. This, however, cannot be accepted as conclusive evidence that the convulsion was dependent upon the worm, as I have met with instances in which large numbers of these worms were found at the autopsy in children who during life showed no nervous symptoms whatever. As a rule, the presence of these parasites in the intestine, unless in very large numbers, is not especially important.

TREATMENT.—The most efficacious treatment of this form of parasite is in the form of santonin, which should be given in the same doses and with the same caution as I have already described in speaking of the treatment of the oxyuris. Instead of santonin, the freshly prepared fluid extract of spigelia and senna in doses of 1.87 c.c. (½ drachm) for a child two years old, and 3.75 c.c. (1 drachm) for older children, can be given two or three times a day, care being taken not to produce too much irritation. The oil of chenopodium, 0.012 to 0.018 c.c. (2 to 3 minims), on sugar, for a child two or three years old, and 0.50 to 0.60 c.c. (8 or 10 minims) for older children, can also be given. A cathartic should be used in connection with these drugs, as well as with santonin.

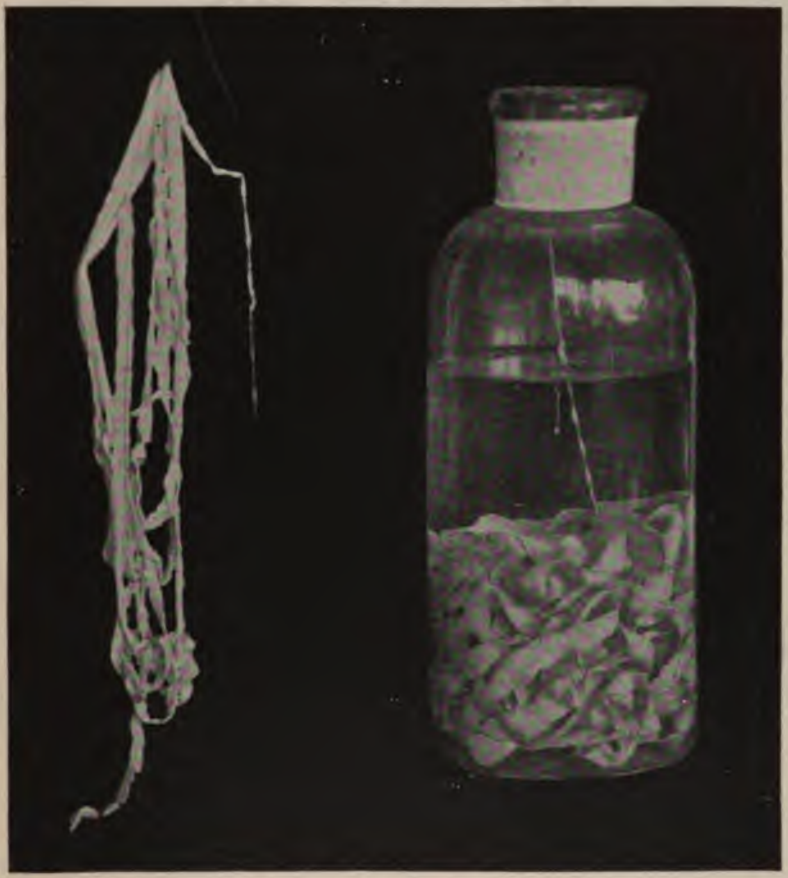
Tæniæ (*tapeworms*).—Two forms of tænia occur in children. One of these is the *tænia solium*, the pork tapeworm. It has a slight projection at the apex of its head, around which are a series of hooks, and below which are four sucking-disks. The other form is the *tænia mediocanellata*, the beef tapeworm. It has a blunter hook than the tænia solium, and does not have the circle of hooks. These worms vary in length from 605 to 1512.5 cm. (20 to 50 feet). There is nothing especial to be said concerning these worms, and I refer to them merely because at times they occur in early life. They are never met with in nursing children when breast-milk forms the exclusive diet. There is reason to suppose that raw-beef juice may carry the eggs of the tænia. There are no especial symptoms produced by this worm, and the diagnosis is made entirely by finding the segments in the fæces. There is no especial danger from the presence of the tapeworm.

TREATMENT.—The treatment employed for expelling this worm is the same in children as in adults, but we should be very careful not to irritate too much the sensitive gastro-enteric mucous membrane of the young child. The child should first be treated with laxatives, so as to free the intestine. Food should be withheld from the early evening until as late as possible the next day. An anthelmintic should then be given, followed in one or two hours by a cathartic. This usually results in the expulsion of a large mass of segments. Great care should be taken to prevent the head from breaking off before it is expelled. If the head remains, the worm grows again and the treatment has been useless. The anus should be carefully dilated during the expulsion of the worm. Sitting on a vessel of hot water seems to help to prevent the head from breaking off.

There is no anthelmintic which I have found especially successful in expelling the tæniæ. One of the most harmless is the alkaloid pelletierine from pomegranate. The tannate of pelletierine can be given to a child from three to five years old in the dose of from 0.01 to 0.03 gramme (⅙ to ½ grain). As dizziness and headache are sometimes complained of, it is well to have the child kept in bed and lying down until the effect of the anthelmintic has passed off. The oleoresin of male fern may also be used. The dose is 0.94 to 1.88 gramme (¼ to ½ drachm). The cathartic which is most useful in these cases is Epsom salt, 7.5 to 15 grammes (2 to 4 drachms).

It is hardly worth while to mention the other numerous anthelmintics which have been recommended, as they are usually inefficient.

Fig. 179.



Tænia. I., without head; II., with head.

If the head of the worm is not obtained and if a considerable portion of the segments is discharged as a result of the treatment, it is desirable to wait a few months before making a second attempt to dispel the parasite. The reappearance of segments in the stools indicates that the growth of the worm is sufficient to justify another attempt to dislodge it.

DIVISION XIII.

DISEASES OF THE LIVER, PANCREAS, SPLEEN, AND PERITONEUM.

DISEASES OF THE LIVER.

THE size of the liver is in infancy very large relatively, and in early childhood somewhat larger than at a later period of life. This is of importance in making a diagnosis as to the increase or decrease of the normal hepatic area. These changes, although varying so much at different ages from infancy to puberty, yet in the intermediate extremes of size show such slight differences that nothing definite can be stated in regard to them. In early life, however, we may say that the edge of the liver may be felt about 1.5 to 2 cm. (½ to ¾ inch) below the edge of the ribs in front, and that the hepatic dulness extends up to the fifth interspace or rib in front, to the seventh interspace in the axillary line, and to the ninth space or rib behind.

The left lobe of the liver is relatively larger in the child than in the adult. (Steffen.) In determining, however, whether or not the liver is of abnormal size we must consider that a downward displacement of a normal liver may take place from relaxation of the ligaments and of the abdominal walls in cases of wasting disease, also from pressure, as from a pleuritic effusion or from thoracic deformity as in rhachitis. Upward displacement also may take place from ascites and other causes of abdominal distention.

Diseases of the liver are not common in infancy and childhood, as the inciting causes of hepatic disease are usually not present in early life. When hepatic disease is present, it is commonly secondary to some general disease.

Congenital malpositions and malformations of the liver may occur, but those of especial clinical interest are chiefly connected with the bile-ducts, and have been described on page 308.

The acquired pathological lesions which occur in the liver in infancy and childhood do not differ from those which are met with in later life. A rapid increase and decrease in the size of the liver are not infrequently met with in disease, and careful measurements have shown that even a very slight disturbance of health may cause in young children a variation of from 2 to 4 cm. (¾ to 1½ inches) in the size of the liver.

In most diseases which are accompanied by hepatic disturbance, it is much more common to have the liver enlarged than diminished in size.

ICTERUS.

Icterus is a symptom of a number of diseases, as well as of disease of the liver, but it so commonly occurs when the liver is either directly or indirectly affected that it is best spoken of in connection with hepatic disease. The icterus which arises at birth, either of the temporary form, such as icterus neonatorum, or from obliteration of the bile-ducts, has already been described on page 308, but it may be added to what was said regarding the former that there is some reason to suppose that it may be caused by the ductus venosus remaining patent at birth. Icterus as a symptom of acute and chronic duodenal indigestion has been described on page 799. It must not be assumed that there is necessarily hepatic disease because icterus is present, as any slight mechanical disturbance in the liver produced by diseased conditions elsewhere may cause icterus. In these cases, even though the liver may be somewhat enlarged, it is not a symptom of much import, and the liver is soon restored to its normal condition, provided that the original disease has disappeared or has ceased to produce hepatic disturbance. In addition to this obstructive class of cases, icterus may also occur as a symptom in *septic inflammation of the umbilical vein.* In these cases the liver is apt to be enlarged and tender. Convulsions commonly occur. Vomiting, diarrhœa, abdominal swelling, pain, and tenderness are present. The temperature is high. The respirations are increased, and death usually occurs from exhaustion or from septic inflammation of the pleura, pericardium, or other parts.

ACUTE YELLOW ATROPHY OF THE LIVER.

It is uncommon for the liver to be decreased in size, but this occurs in the very rare cases of acute yellow atrophy at times met with in children. The disease is insidious in its onset, and is characterized by general symptoms of malaise, with icterus and urine containing bile. In the beginning of the disease the liver is enlarged, but in the later stages it is decidedly diminished. Cerebral symptoms and vomiting are quite prominent, and death invariably occurs.

CONGESTION OF THE LIVER.

Although an acute congestion of the liver may occur, as from certain poisons and in the course of malaria, yet by far the most common form is the subacute or chronic condition which results from stasis in the venous system, produced by cardiac disease and certain pulmonary conditions which interfere with the circulation.

There are no especial hepatic symptoms beyond enlargement of the organ, and the treatment is that of the primary disease. This enlargement may occur from a number of causes, among which is *mechanical congestion*, arising in the course of cardiac disease.

FATTY INFILTRATION OF THE LIVER.

Fatty liver in early life is quite common, especially in infants, and does not differ pathologically from that which is met with at a later period. The liver may or may not be enlarged, and there are no especial hepatic symptoms, such as icterus or ascites, which characterize this condition, the symptoms being those of the general disease from which the child is suffering. It may be found associated with a number of diseases, especially rhachitis, tuberculosis, and wasting diseases, especially with those which are associated with disturbances of digestion. When the liver is enlarged from this cause its surface is found to be smooth and there is no pain nor tenderness on palpation.

The prognosis, unless the disease is dependent upon some incurable disease elsewhere, is fairly good.

The treatment is essentially dietetic, hygienic, and that of the original disease.

SUPPURATIVE HEPATITIS.—ABSCESS OF THE LIVER.

Abscess of the liver is an exceedingly rare condition in both infants and children. Even when septic infection of the umbilical vessels has taken place at birth, it has been found that only in a very small proportion of these cases has hepatic abscess resulted.

ETIOLOGY.—According to Musser's analysis of thirty-four reported cases, the average age was nine years, and the youngest one year. The causes in these cases were from traumatism, from round-worms entering the bile-ducts, from pylephlebitis in four cases, from umbilical phlebitis once, from pyæmia twice, and once each from pelvic peritonitis, dysentery, perityphlitis, malaria, and tuberculosis of the lungs. Musser therefore concludes that the general conditions, such as climate and habits of life, which are marked etiological factors in adults, are not of significance in children.

SYMPTOMS.—The onset, the progress, and the duration of the disease depend greatly on the primary cause. Icterus may occur, but usually is not marked, and is not of much significance. The local symptoms connected with the liver are the same as in adult life, and are chiefly pain, not always, however, in the hepatic region, but in different parts of the abdomen, enlargement of the liver, usually downward, and tenderness over the liver. If in addition to these symptoms a tumor is detected apparently connected with the liver, and accompanied by fever of a hectic, pyæmic, or intermittent type, with sometimes an initial chill or with irregular daily chills, abscess of the liver is to be strongly suspected. Leucocytosis is present. The symptoms are at times, however, very latent, and the diagnosis can only be determined by aspiration.

The duration of the disease is apt to be prolonged, and the child gradually emaciates and fails in strength.

The prognosis varies greatly, as there are so many conditions as to the especial cause and the especial locality of the abscess which must be taken into account and which render the chances of recovery more or less uncertain.

TREATMENT.—The treatment is essentially surgical. Musser reports twelve recoveries out of the thirty-four cases, and in eleven of these aspiration or incision or both were employed.

HYDATIDS.—BILIARY CALCULI.—NEW GROWTHS.

Echinococcus cysts and biliary calculi are so rare in early life that they need merely be mentioned as of possible occurrence. Carcinoma, adenoma, and sarcoma of the liver have in rare instances been met with in early life. Tuberculosis of the liver has been described on page 431.

AMYLOID LIVER.

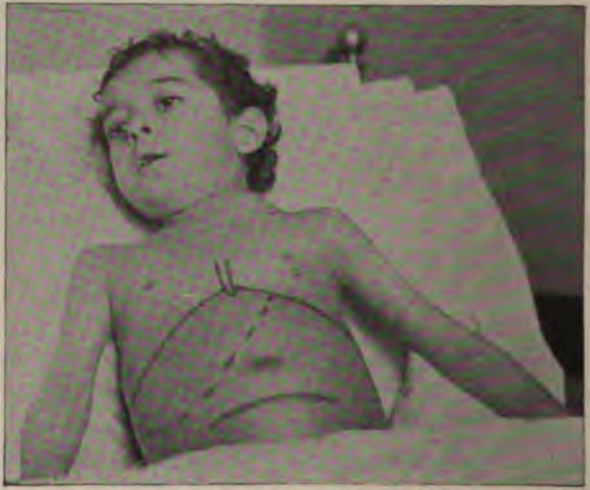
When amyloid changes are present in the liver, other organs, such as the spleen, kidneys, and intestine, are involved. Amyloid infiltration may occur in the course of tuberculosis when there is chronic disease of the bones with extensive suppuration, in wasting diseases, and in hereditary syphilis. A very prominent symptom in this condition is an extreme degree of secondary anæmia. The liver is, as a rule, very much enlarged, and commonly more so than in any of the other hepatic disturbances. Its surface is smooth, and there is rarely hepatic tenderness or pain. Ascites is rare, and there is usually no icterus. The progress of the disease is slow.

The diagnosis is not difficult if we find that the child has one of the diseases which have just been mentioned as being the causes of amyloid changes. The skin has a waxy appearance. The spleen is enlarged, and there may be albuminuria and dropsy, either from an associated amyloid condition of the kidney or from the pressure produced by the enlarged liver.

When these changes occur in the liver the prognosis is very grave, and there is no treatment which will be of more than temporary benefit. The treatment, therefore, is removal, if possible, of the cause, such as a diseased joint. If the disease is the result of syphilis, anti-syphilitic remedies should be employed.

Fig. 180 represents a boy seven and three-quarter years old. There was no history of tuberculosis in his family. He had pertussis when he was one and a quarter years old, and measles when he was three years old. He seemed well and strong until he was about seven years old, when he became listless and began to have fever and to perspire profusely. Later he began to vomit occasionally, to complain of headache, and to cough. Although he evidently lost in weight he was not especially emaciated. His entire skin was extremely pale and had a waxy look, which was apparently not due to jaundice. His mucous membranes showed much anæmia. His tongue was heavily coated, and his breath was offensive. He was dull and apathetic. The cervical glands were enlarged and slightly tender, but did not fluctuate. The glands were moderately enlarged in the axillæ and groins. The percussion of the right lung, especially at the apex, was dull, and there were numerous râles. The area of cardiac dulness was not enlarged, but there was a slight systolic murmur at the apex. The spleen was slightly enlarged. The edge of the liver could be felt below the line of the umbilicus. The area of hepatic dulness was increased, as is represented by the broken line in the figure. There was no hepatic tenderness, and the child did not complain of pain. The lower part of the abdomen was dull on percussion as high as the line which is drawn under the umbilicus. This was due to a slight amount of ascites. The legs were swollen. The urine had a specific gravity of 1010, and contained a slight trace of albumin, an occasional hyaline cast, and renal epithelium. The temperature had varied from 37.2° to 39.4° and 40.5° C. (99° to 103° and 105° F.). The increased size of the liver was probably due to amyloid infiltration, following what was supposed to be pulmonary tuberculosis.

FIG. 180



Amyloid liver. Pulmonary tuberculosis. Male, 7¾ years old.

A few days later the child grew rapidly weaker, and died of exhaustion. An autopsy was not obtained.

INTERSTITIAL HEPATITIS.—CIRRHOSIS.

Interstitial hepatitis may be a lesion of a number of systemic diseases, especially of syphilis, but also of tuberculosis. The disease, however, may apparently occur without disease of any other organ. In infants cirrhosis of the liver is almost invariably of syphilitic origin.

A certain number of cases seem to have followed scarlet fever and measles. Alcohol is sometimes an etiological factor in infancy and early childhood. When the disease is caused by alcohol the pathological condition is, as a rule, atrophy.

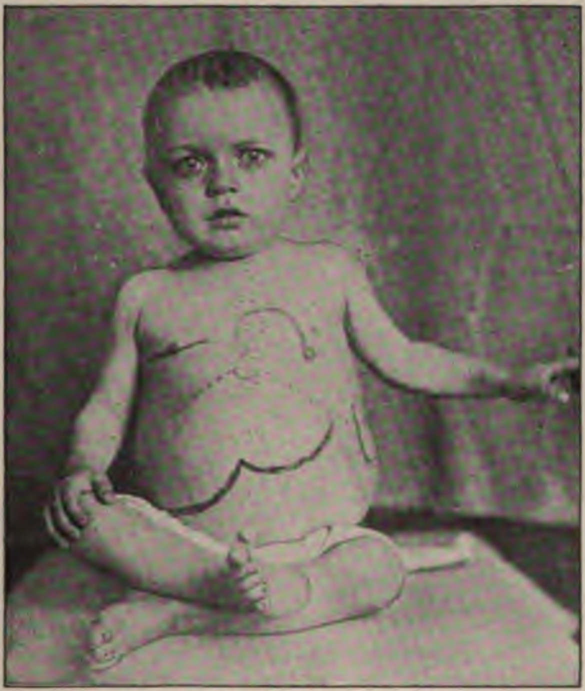
Interstitial hepatitis as it occurs in childhood may be *atrophic* or *hypertrophic*. The general symptomatology differs but little from that of the adult. In the beginning the symptoms are very apt to be confounded with those of simple congestion arising from digestive disturbances. There may be abdominal pain, slightly augmented by pressure. Diarrhœa and constipation alternate. There are usually ascites and slight jaundice, and at times dilatation of the subcutaneous abdominal veins. Stigmata composed of collections of dilated minute veins are sometimes observed on the face. The temperature is irregular. As a rule, it is not much heightened, and, in fact, is often subnormal.

Enlargement is not common, and the symptoms are the same as in the adult, the ascites being especially prominent.

PROGNOSIS.—The prognosis in this class of cases is bad, as the progress of the disease is usually rather rapid.

When the hepatitis is not dependent on disease elsewhere, and is not due to alcohol, there are no characteristic symptoms beyond the enlargement of the liver. In this form the ascites is usually small in amount, and the diagnosis can be made only by eliminating the other forms of enlargement. It is commonly spoken of as *hypertrophic cirrhosis*. Its prognosis is more favorable than that of the cirrhosis just described. Its treatment is symptomatic.

FIG. 181.



Hypertrophic cirrhosis. Female, 18 months old.

Fig. 181 represents a girl eighteen months old. There was no history of syphilis or of tuberculosis. She had pertussis when she was ten months old, and the cough lasted for several months. She had never been given alcohol in any form. She was well until she was fifteen months old, when she began to complain of pain in the abdomen and to become pale. Before entering the hospital she had diarrhœa, and her abdomen was swollen. On entering the hospital and being placed on a proper diet, the diarrhœa ceased, but the swelling of the abdomen increased. She was not especially thin, but was pale. The abdomen was much enlarged. The edge of the liver could be felt nearly as low as the line of the umbilicus. The area of dulness on percussion is marked by black lines. The lower one shows the notch between the right and the left lobe, which was distinct and easily palpable. There was no especial tenderness on pressure. The spleen was slightly enlarged. In the lower part of the abdomen there was a moderate amount of dulness and fluctuation, showing the presence of fluid. There were no glandular swellings. The heart was normal, but was pushed up somewhat by the abdominal distention.

The child improved in its general health while in the hospital, and had a fair appetite. Physical examination showed the presence of no other disease. Without an autopsy, however, the diagnosis was necessarily held in abeyance.

The child remained in the hospital for a few weeks, and improved in its general health so that it seemed quite bright. The ascites did not increase in amount, but the liver remained enlarged. The child was eventually taken away from the hospital, and its subsequent history could not be obtained.

DISEASES OF THE PANCREAS.

Diseases of the pancreas are practically unknown in infancy and childhood, with the exception of the general tissue-changes which may be met with in syphilis, and which have already been described. New growths of a malignant nature have been reported.

DISEASES OF THE SPLEEN.

The spleen may be involved in tuberculosis, and may show amyloid changes in connection with other organs. It is frequently enlarged in the course of a number of diseases which have been described elsewhere, but there are no primary diseases of the spleen.

DISEASES OF THE PERITONEUM.

Diseases of the peritoneum may be of non-inflammatory or inflammatory origin.

The *non-inflammatory* diseases are mostly represented by new growths. These may be of a malignant nature, such as carcinoma and sarcoma, or they may be lipomata or of a cystic character. Tumors of the omentum are rare, but cysts and hydatids may occur in this region. The differential diagnosis of these various forms of peritoneal and omental growths can scarcely be made during life. The treatment is essentially surgical.

The *inflammatory* diseases of the peritoneum are represented by peritonitis.

ACUTE PERITONITIS.

Etiology.—Acute inflammation of the peritoneum may be primary or secondary. It is a condition of great importance in infancy and early life.

Infants and children of any age may be attacked. The disease may occur in the course of tuberculosis, of the infectious diseases, of syphilis, and, most frequently of all, of appendicitis. The disease in any of the above forms is exceedingly rare between the ages of six weeks and two years.

A large variety of *micro-organisms* are found in connection with acute peritonitis. The colon bacillus is the most frequent cause. Streptococci, staphylococci, and pneumococci are sometimes found either alone or in mixed cultures. In the rare instances of perforation of typhoid ulcers typhoid bacilli may be cultivated from the abdominal fluid in conjunction with the colon bacillus and other intestinal micro-organisms. Tubercular peritonitis has been described on page 401.

Perforation of some viscus into the peritoneal cavity is the underlying cause of many cases of peritonitis. Such an accident may occur from intestinal ulcers, from abscesses and cysts of the liver, gall-bladder, spleen, kidney, lymph-glands, and appendix, and from strangulated hernias and intussusceptions.

Peritonitis may also be set up by *extension* from neighboring inflammatory processes, even when no direct communication has been made with the peritoneal cavity by means of perforation.

Traumatic peritonitis may develop from blows, punctured wounds, and septic infection from surgical operations.

PATHOLOGY.—The pathological lesions in acute peritonitis are represented by reddening and loss of the normal glistening appearance of the peritoneum, soon followed by an exudation varying from a thin serum with a small number of white and red blood-corpuscles to a thick, fibrinopurulent exudate. The character of the pathological processes have been more fully described under the various forms of appendicitis with peritonitis on page 815. In the peritonitis of the new-born the characteristic lesions are apt to be in the umbilical region, and are not infrequently associated with lesions of a general septicæmia.

SYMPTOMS.—The symptoms of acute peritonitis vary according as the process is general or localized. The localized form of peritonitis corresponds in its symptoms to what has already been described in speaking of appendicitis, which is its most frequent cause. In general peritonitis the symptoms in infants are often obscure. In children the symptoms are usually pronounced and characteristic. The child is attacked with abdominal pain and with general abdominal tenderness. The abdomen becomes distended and tympanitic, and the child assumes the position which will most relax the abdominal walls,—that is, with the thighs flexed and the knees bent. Vomiting is very apt to be present, and is augmented when food is given. The bowels are often constipated, although at times there may be diarrhœa. The temperature is usually high, 38.3° to 40.5° C. (101° to 105° F.); in some cases, however, the temperature may be normal or subnormal. The pulse is small and rapid. The respirations are not only accelerated, but also superficial, as deep respiration causes pain. The face has an anxious expression, and shows great suffering. When recovery takes place, these symptoms gradually subside after a few days, the tenderness, pain, and tympanites disappear, and the child's face assumes a tranquil look. When improvement does not take place, the pulse becomes weaker and more rapid, the breathing more superficial and more frequent, there is chilling of the extremities, and the child dies usually within a week. Leucocytosis is generally marked unless the severity of the infection is so great as to render a reaction impossible.

Peritonitis of the New-Born.—This form generally appears within the first two or three days of life, rarely later than the first week. The onset is sudden, with vomiting, diarrhœa, pain, distention of the abdomen, and tenderness. The temperature is very high, 40° C. to 41° C. (104° F. to 106° F.). In a case reported by Quinquand it reached 42.5° C. (108.5° F.). Emaciation is rapid and the cachexia is extreme. Hydrocele, generally of the right side, with œdema of the scrotum, frequently develops. Septicæmia and pyæmia may occur as complications. Death takes place in four or five days.

Acute Pneumococcus Peritonitis.—Peritonitis may be due to a primary idiopathic infection by the pneumococcus, or to a secondary infection from the same organism in connection with pneumonia, pleurisy, or meningitis. Netter has reported a case of pneumococcus meningitis with pneumococcus peritonitis in an infant three or four days old. According to Comby, primary pneumococcus peritonitis is more frequent in children than in adults, and is most common between the ages of three and twelve years. In Brun's series of cases it was nearly four times more frequent in girls than in boys.

Dieulafoy describes pneumococcus peritonitis as a special clinical form, which can sometimes be diagnosticated by the character of the symptoms.

SYMPTOMS.—The onset is sudden, with intense abdominal pain, diarrhœa, and fæcal vomiting. The temperature rises to 39° to 40° C. (102.2° to 104° F.), and there may be delirium. A remission of some of these symptoms then ensues, the pain subsides, diarrhœa and distention persist, and the picture is more like that of typhoid fever. At the end of about eight days the temperature falls, sometimes as abruptly as in pneumococcus pneumonia. The disease then passes into another stage, which resembles tubercular peritonitis. There is emaciation, cachexia, pinched features, sunken eyes, and a large and tender abdomen, usually with signs of fluid. The fluid, which is purulent, is not very movable, but shows a strong tendency to become encysted. The process may go on for several weeks and finally end in spontaneous evacuation of the pus. While recovery may take place by natural means, the danger of secondary infection and death from exhaustion is so great that early surgical interference is indicated, as under favorable conditions a good proportion of the operative cases recover.

Prognosis.—The prognosis in acute peritonitis is always grave. It depends largely upon the virulence of the exciting cause, upon an early diagnosis, and upon prompt and skilful surgical treatment.

Treatment.—The treatment is essentially the same as has been described under appendicitis with peritonitis on page 819.

The following case illustrates acute peritonitis:

An infant, nineteen months old, previously apparently healthy, was attacked with vomiting and diarrhœa. On the following day the face was pale, the alæ nasi were working slightly, the respirations were 36, and the temperature was 39.4° C. (103° F.). The respirations gradually increased to 74, and the temperature rose to 40.3° C. (104.6° F.). The abdomen became very much distended and tender, and the face pinched and anxious. On the evening of the second day from the onset of the disease the temperature rose to 41.1° C. (106° F.), the infant became very restless, the pupils were contracted, and death took place a few hours later.

The autopsy showed that the heart and lungs were normal. The spleen was enlarged, and was covered with a fibrinous exudation. The kidneys were pale, and normal in size. The liver was covered with flakes of lymph of recent formation, and on section showed the acini to be red and their periphery yellowish and opaque. The mesenteric lymph-nodes were slightly enlarged, and the smaller ones were translucent on section and presented evidence of hyperplasia. A small pocket of the larger nodes was found to have become cheesy in the central portions, and in two of these the process had extended through the substance of the gland and had broken through its peritoneal covering. About these points of rupture there was a small zone of reactive inflammation.

The pathological diagnosis was acute general peritonitis, which, from an absence of any other source, must be considered to have been caused by the rupture of the cheesy, degenerated mesenteric nodes.

In this case the high temperature and the distended abdomen rendered the diagnosis comparatively clear. The case is important on account of the cause, for there is seldom any noticeable enlargement of the mesenteric nodes under the age of three years, and these nodes seldom soften, but either retrograde or harden from calcification.

CHRONIC PERITONITIS.

By far the most common cause for chronic peritonitis with ascites is tuberculosis, which has been described on page 401.

When an acute peritonitis is localized, the inflammatory process may subside, the fibrino-purulent exudation may become organized, and the result be fibrous adhesions binding the coils of the intestine closely together and to the abdominal wall.

A form known as *congenital peritonitis* occurs, but is of pathological rather than of clinical interest. In this form the fibrous adhesions are found in infants who have died within a few hours after birth. It is considered a peritonitis of intra-uterine development, possibly through the placenta from an intra-uterine peritonitis in the mother.

DIVISION XIV.

DISEASES OF THE KIDNEYS, BLADDER, AND GENITAL ORGANS.

DISEASES OF THE KIDNEYS.

Diseases of the kidneys may be congenital or acquired.

CONGENITAL DISEASES.

The congenital abnormalities, such as congenital cystic kidney, absence of one kidney, hypertrophy of the remaining kidney when one is absent, anomalous shapes of the kidney, and malpositions of the ureters, are important, but are of surgical rather than of medical interest. The lobulated kidney, already described as a normal condition in intra-uterine life, may to a greater or less degree continue into infancy and childhood, but has no pathological significance. Movable kidneys are rare in early life, but have been reported.

ACQUIRED DISEASES.

Renal disease as a primary affection in infancy and childhood has been considered rare, but this view has been modified by later investigations, which have shown that nephritis is not uncommon in cases of general infection. Secondary renal lesions are comparatively common.

The systematic examination of the urine in children in connection with the different pathological lesions of the kidney has not yet been made to such an extent that we can invariably differentiate the especial type of lesion by the character of the urine. Clinically, the diseases of the kidney, as differentiated by the urinary examination in infancy and early childhood, are less varied than in adults. They are represented chiefly by active hyperæmia and the mild and severe forms of acute nephritis following scarlet fever especially and other of the acute infectious diseases.

General Etiology and Pathology.—According to Councilman, to whom I am indebted for much information on this subject, the acquired diseases of the kidney in childhood show considerable differences from the renal diseases of the adult. In childhood there is a greater liability to those acute affections, such as scarlet fever, measles, and diphtheria, in the course of which nephritis is apt to appear. Children under the age of fifteen years are less subject to many pathological conditions, such as disorders of the circulation, which in the adult frequently lead to chronic lesions of the kidney. Children do not usually have those disorders of the circulation which result in granular kidney, for lesions of the arteries, especially the condition known as arterio-sclerosis, do not commonly occur in childhood. While it is true that typical examples of the small granular kidney are sometimes met with in children, these lesions of the kidney are primary, and the lesions of the circulatory system are secondary and dependent on the renal lesions. A part of the chronic diseases of the kidney in the adult is without doubt to be referred to the continuous action on the kidney of slight pathological conditions, an action from which the child's age protects it. One pathological lesion not perfectly recovered from, moreover, makes the kidney more prone to disease, and a greater effect will be produced a second time by the same cause, and chronic disease may result. In the kidney of the adult, with the advance of years there is a gradual decline in the power of regeneration, and slight troubles are not readily recovered from. The kidney of the child, on the other hand, is an organ which possesses great power of growth and regeneration. For this reason a condition which in the adult organ is either not recovered from at all, or lays the foundation for chronic disease, may in childhood result in complete recovery. Again, the child is not exposed to certain conditions which are productive of chronic lesions, or which may lay the foundation for them. Among these may be mentioned alcoholism and excesses of various sorts. Many cases of nephritis in the adult are to be referred to causes acting not through the blood, but through the urinary tract. The child, on the other hand, is not exposed to the dangers arising from hydronephrosis and pyelonephritis, except to a very limited degree. Although the causes of renal disease are less numerous and less common in children than in adults, yet when the same etiological factor is present the same morbid condition is produced in the kidney. The various cachectic conditions will lead to amyloid infiltration in the child as they do in the adult, and amyloid infiltration of the kidney makes up by far the larger part of the chronic cases of albuminuria in children. We may also meet with certain chronic lesions in the child's kidney, such as are seen in tuberculosis, and these may lead to albuminuria and to nephritis.

The acute diseases of the kidney may either lead to recovery or may of themselves prove fatal. They are not to be referred to the continuous action of the poison of the acute disease, but to the effect on the kidney of the lesions produced by the inflammatory process. An example of this is the condition of chronic nephritis after scarlet fever, in which the acute lesions gradually pass into the chronic. These chronic lesions are to be attributed to the disorders in the circulation of the organ brought about by the destruction of the glomeruli.

GENERAL SYMPTOMATOLOGY.—The general symptoms connected with the various forms of nephritis are so similar that it will be less confusing to mention first the common symptoms which may occur in any of the forms of nephritis, and then to describe the etiology, pathology, and urinary examination of the different forms.

One of the most common signs in nephritis is œdema, which occurs frequently in acute nephritis and in chronic parenchymatous nephritis. The œdema generally appears first in the eyelids, and then in the hands and feet. There may be general anasarca. Not infrequently, however, œdema is absent or not marked. Nausea and vomiting are not infrequent in the beginning of the disease, and in some cases are, perhaps, due to the heightened temperature. It may occur later in the disease as a symptom of uræmic poisoning. In such cases there is marked diminution in the amount of the urine, or even suppression. A marked secondary anæmia giving a peculiar dull white color to the skin is commonly seen in chronic parenchymatous nephritis, and is quite striking. In acute nephritis fever is often present to a greater or less extent, but is a variable symptom. Lack of appetite, and weakness, are common in both acute and chronic nephritis. Headache is a variable symptom. It is a frequent symptom of uræmia, and sometimes the only one. Restlessness and insomnia are often met with, and attacks of dyspnœa may occur. Amaurosis may occur as the result of albuminuric retinitis, or it may be a functional symptom of the uræmic poisoning and disappear later if the patient recovers. Hypertrophy of the left ventricle with a pulse of high tension is apt to occur in interstitial and chronic parenchymatous nephritis. Both diseases are, however, very uncommon in childhood. In acute nephritis following scarlet fever dilatation and moderate hypertrophy of the left ventricle are not uncommon. Transudation into the serous cavities has been reported in a number of cases, as has also œdema of the larynx.

In all cases of nephritis the amount of urea excreted during the twenty-four hours should be carefully estimated from time to time, as a decrease in the urea always suggests a pathological condition, and a return to the normal amount is usually indicative of recovery unless there is a complication with some other disease. Any interference with metabolism, however, whether in the liver or in the lung, may diminish the amount of urea in the urine. In children during convalescence from acute nephritis the urea returns to or exceeds the normal amount, while in chronic nephritis it is always diminished, as it is in adults. A sudden and excessive diminution of the urea in acute nephritis is suggestive of uræmia. In acute and chronic nephritis the chlorides are diminished when an effusion such as ascites is increasing, and gradually return to the normal amount as the effusion is absorbed.

In any attempt to estimate the functional activity of the kidney, either as regards the quantity of urine or the amount of solids excreted, it is very desirable to know the amount passed in twenty-four hours. This is practically impossible in infants and very young children, in whom it is often difficult to obtain even a single specimen. Various methods have been devised for collecting the urine in infants. The most simple and effective is a cotton ring large enough to enclose the buttocks and genitals. The ring is lined with sheet rubber, and the infant (the napkin having been removed) rests upon it comfortably, and the urine can be collected as in a bed-pan.

ANURIA.

Anuria is an arrest of the secretion of urine. It must be distinguished from retention of urine in the bladder, from the scanty urine passed when liquids are withheld or refused in sickness, and from diminished secretion dependent upon a profuse watery diarrhœa.

Total suppression of urine may occur in the intense congestion of acute nephritis, in poisoning by lead, phosphorus, or turpentine, or after severe injuries or operations.

Anuria in the new-born may be due to malformation of the urinary tract or to uric acid infarction. Other cases in infants are evidently of nervous origin, and may last for twenty-four or thirty-six hours without other symptoms. Hysterical anuria may occur in older children. Charcot reports a case lasting for eleven days.

TREATMENT.—The obstructive cases demand operation. In non-obstructive cases the treatment is usually simple and satisfactory. Sweet spirits of nitre or citrate of potash given with plenty of water will usually relieve the condition. Hot applications over the kidney may be used, or large hot irrigations of the colon with salt solution.

PHYSIOLOGICAL ALBUMINURIA.

This condition is not infrequent, and may occur at any period of infancy and childhood, but is most common between the fifth year and puberty. It is more common in boys than in girls. The amount of albumin present is, as a rule, less than one-twelfth per cent., and is sometimes associated with temporary glycosuria. It is not present in every micturition, and in many cases seems to depend upon over-exercise, cold bathing, or a highly nitrogenous diet. The albumin is rarely present in the urine which is passed in the morning immediately after rising, and this is an important point in differentiating physiological albuminuria from periodic albuminuria due to pathological causes, such as uric acid. The presence of blood-corpuscles or abnormal elements in any amount from the kidney shows that there is a pathological condition. The children who have this physiological albuminuria often seem to be in good health, but sometimes they are rather delicate. The diagnosis can be made only by repeated examinations of the urine passed at different times in the day, and by observing the effect of exercise and diet upon it. An occasional hyaline cast and albumin as high as one-fourth per cent. for short intervals may be present. The albumin often disappears for a time and returns again.

Albuminuria of Adolescence.—One form of physiological albuminuria is that which has been called the albuminuria of adolescence. At puberty there appears to be a disturbance of the equilibrium of the renal circulation, occurring so frequently, and presenting so distinctively the characteristics of a simple hyperæmia, that we are justified in looking upon it as a physiological rather than as a pathological condition.

This physiological congestion of the kidney is probably closely connected with the development and increased activity of the uterine circulation in the female, and with the prostatic and genital blood-supply in the male. The importance not only of knowing that such a condition exists at puberty, but also of bearing it in mind when we are called to treat children who are on the border-line between childhood and adolescence, is too little recognized, and this want of recognition often leads to unfortunate mistakes.

Prognosis.—The prognosis in these cases of physiological albuminuria is good, and, so far as I know, no cases have been reported in which the condition terminated in nephritis.

Treatment.—The treatment is to regulate the diet, exercise, and general hygiene carefully. If the children are anæmic, iron is indicated.

The following is one of a number of cases of albuminuria of adolescence which have come under my notice:

A girl, thirteen years old, was brought to me for advice with the following history. She had always been somewhat delicate, but had never had any especial disease, and was considered to be fairly healthy, until she was twelve years old. She then began to grow very fast in height without a corresponding development in weight and general muscular strength. When she was twelve and a half years old the catamenia appeared, and were accompanied by severe pain. Six weeks later, the catamenia again appeared, and were accompanied by considerable pain and general prostration. The child at this time looked pale and thin, had very little appetite, and was easily fatigued. A physician was consulted, who prescribed strong food, such as meat, a tonic, and gymnasium exercise. This advice was followed implicitly, and the child was made to exercise especially the muscles connected with the abdomen and pelvis three or four times a week at the gymnasium, and by daily home exercise, such as lying on the back and raising the legs. Under this treatment the child rapidly grew worse, and the catamenia did not return in the following month.

A physical examination of the child showed nothing abnormal beyond too rapid growth. The total quantity of urine in twenty-four hours was 960 c.c. (32 ounces). The color was normal, reaction acid, specific gravity 1023, urea increased, a very slight trace of albumin, sugar absent. The sediment showed a considerable amount of mucus, a little vaginal epithelium, an occasional granular and epithelial cast, renal epithelium, and an occasional blood-globule.

The urine was simply one which showed a slight renal hyperæmia, and the child was therefore treated as follows. She was not allowed to go to school or to the gymnasium, but was made to rest in bed for several hours twice a day. Her diet was largely milk in considerable quantity, meat especially being withheld. She was also made to drink freshly distilled water, 250 c.c. (about eight ounces) once in six hours. She was allowed to take a slight amount of exercise out of doors, but to a very limited degree.

Under this course of treatment the child began slowly to improve. She became less anæmic; her appetite increased, and was less capricious; she began to gain in weight, to sleep well, and to have more strength.

Subsequent examinations of the urine showed a progressive improvement until, at the end of one year, the albumin had entirely disappeared and the urine became normal.

HÆMATURIA AND HÆMOGLOBINURIA.

Hæmaturia is the term applied to the presence of red blood-corpuscles in the urine. Hæmoglobinuria is the term used to describe those cases in which the hæmoglobin or blood-pigment is present in the urine without the red blood-corpuscles themselves. Both conditions are to be considered as symptoms of some primary disease rather than distinct diseases in themselves. Hæmaturia and hæmoglobinuria are, as a rule, easily recognized by the color of the urine if sufficient blood or blood-pigment is present to color it. The color is red if it is due to fresh blood, or brownish red if due to blood-pigment which has been washed out of the corpuscles.

Etiology.—Hæmaturia is often the first indication of an acute nephritis from any cause. It may also occur as a result of scorbutus, hæmophilia, pernicious anæmia, and leukæmia. Malignant disease of the kidney, uric acid concretions in the kidney and bladder, and trauma, are at times the direct cause of the condition.

Both hæmaturia and hæmoglobinuria, usually in small amounts, may be caused by poisons, such as carbolic acid, cantharides, and chlorate of potash, and also by infectious diseases, such as scarlet fever, typhoid, and malaria.

Paroxysmal hæmoglobinuria is of rare occurrence in childhood. The cause is obscure. Some cases are probably of malarial origin; many others seem to be dependent upon over-exertion and exposure to cold, such as may come from chilled or wet feet, or from a cold-water plunge.

Symptoms.—With the onset of the attack there may be severe symptoms of chills, fever, rapid and small pulse, and prostration. Again there may be hardly any subjective symptoms, the color of the urine alone being noticed. When the hæmaturia is dependent upon an acute nephritis, the symptoms follow the course of the primary infection.

Diagnosis.—To determine the source and cause of the hemorrhage is often quite difficult, and at times is impossible. When the blood comes from the bladder it may not be uniformly diffused through the urine, and small clots are common. In addition to this there are symptoms of disturbance of the bladder, such as tenesmus and frequent and perhaps interrupted micturition. In hemorrhage from the kidney the blood is diffused through the urine. The color may be red or brownish red. The microscopic examination shows epithelium and casts from the kidney, many of which contain red blood-corpuscles embedded in them, and are known as blood-casts, and the renal elements are stained yellow and brown from long contact with the blood. There are also normal blood-corpuscles, and others from which the hæmoglobin has been washed out, which appear as pale rings, the biconcavity being lost.

The diagnosis of hæmaturia rests upon the detection, by microscopic examination, of the red blood-corpuscles, either in a normal condition, or decolorized, in the urine. When these are absent and the color of the urine suggests the presence of blood-pigment, the hæmin test by glacial acetic acid will show the characteristic crystals if blood-pigment is present.

Infectious hæmoglobinuria has been described on page 314.

Treatment.—The treatment of hæmaturia and hæmoglobinuria consists in that of the primary diseases. The cause should be determined, if possible, and the indications for treatment followed. Rest in bed and a milk diet until the urine is of normal color should be enforced.

CHYLURIA.

Chyluria is a rare disease. Two forms are usually spoken of, the tropical and the non-tropical.

Etiology.—The tropical form is caused by a parasite, the *filaria sanguinis hominis*, a species of round-worm. This parasite is found in the blood during the night; in the daytime the blood is almost or entirely free from them. The parasite is found at times in the urine. The exact connection between the parasite and the chyluria has not yet been determined. In the non-tropical form the parasite has not been found. Cases have been reported in which the parasite appeared in an individual residing in the tropics, and disappeared on his returning to a cold climate, although the chyluria continued. The chyle is supposed to get into the urine after it has left the kidney.

Symptoms.—The symptoms of this disease are shown chiefly in the urine. The urine has a milky appearance, sometimes a sour odor, and tends to decompose rapidly. The reaction is slightly acid, or neutral. Microscopic examination shows the fluid to be filled with fine granules or fat drops in suspension. The urine at times contains blood-corpuscles, and albumin is always present. The attacks are apt to be paroxysmal, lasting for days or weeks, then ceasing and again recurring. A fatty diet may or may not cause an increase in the chyluria. The individuals affected by the disease may have a healthy appearance. Coagula may at times be formed in the bladder and give rise to pain and difficult micturition. The lymph-nodes, especially in the inguinal region, are sometimes enlarged.

Prognosis.—The prognosis of chyluria is doubtful. It is a disease which lasts for a long time, and may cause anæmia and emaciation from the loss of fat and albumin.

Treatment.—There is no drug known which destroys the embryos in the blood. In infected districts the drinking-water should be boiled. A diet containing little liquid and little fat will cause the chyle to disappear from the urine, but this, of course, does not constitute a cure.

The removal of the adult worms from the enlarged lymph-nodes by surgical means has been the most successful treatment.

GLYCOSURIA.

Glycosuria is a symptom, characterized by the transient appearance of sugar in the urine. It may result from poisoning with such substances as carbonic acid gas, morphine, or mercury, or it may appear in connection with acute infectious disease, such as diphtheria, scarlet fever, typhoid fever, or malaria. It is not infrequently dependent upon some disturbance of the nervous system resulting from a fracture of the skull, cerebrospinal meningitis, or epileptiform convulsions. Glycosuria may occur in infants as well as in children, although usually not in those who are healthy. It is often associated with digestive disturbances. It may depend partly on the diet, and in these cases is called *dietetic* or *alimentary glycosuria*, and Koplik found glycosuria in five out of ten infants who were taking a food largely composed of sugar. The amount of sugar excreted is usually small and its appearance transient, by which the condition may be readily distinguished from the disease diabetes mellitus if the urine is examined frequently and at different times during the twenty-four hours.

ACTIVE HYPERÆMIA.

Etiology.—An active hyperæmia of the kidneys may arise in the course of various acute infectious diseases from the elimination of irritating soluble toxins. Fermental diarrhœas and acute ileo-colitis are frequent causes of active hyperæmia in children. It may also be caused by an excess of uric acid, and by such irritating drugs as turpentine, cantharides, and arsenic, lead, large doses of calomel, salicylic acid, and potassium chlorate. Concentrated urines, and urines containing bile and sugar, also act as irritants, and lead to a hyperæmic condition of the kidneys. Irritants due to inflammations of the bladder from gonorrhœal and tubercular infections may, by extension upward along the ureters, start up an active hyperæmia of the straight or collecting tubules. When the action of these causes is very intense, or when the irritation is sufficiently prolonged, the hyperæmia may develop into a true acute nephritis.

Pathology.—The pathological conditions resulting from active hyperæmia of the kidneys are represented by degeneration and desquamation of the renal epithelium and injection of the blood-vessels. There is also to some extent an infiltration of round cells. The process seems to affect chiefly the epithelium of the tubules.

Symptoms.—Unless the hyperæmia is very pronounced, there are, as a rule, no general symptoms.

Urine.—The urine is clear, acid, and its color is often normal. If the cause of the hyperæmia is due to a febrile disturbance, the urine is generally high-colored, concentrated, and diminished. If the cause is not febrile, the urine is often either normal in quantity or dilute. The amount of nephritis corresponds to the condition of urine seen in *active hyperæmia.* (Ogden.)

SYMPTOMS.—The symptoms of acute nephritis are such as have been already described under scarlet fever on page 572. In general, the symptoms arising in cases due to other causes than scarlet fever are the same, but less severe than those described in connection with that disease. Briefly they may be stated to be, sudden onset with mild fever; rapid development of anæmia; swelling of the eyelids, face, ankles, and often general anasarca; headache, thirst, nausea, and vomiting; frequent micturition, but diminished quantity; and in case of the advent of uræmia, stupor and convulsions.

Urine.—The characteristics of the urine are generally described according to three recognizable stages.

First or Acute Stage.—The quantity is very much diminished, at times almost suppressed. The color is dark, smoky, or blood-red. The reaction is acid, rarely alkaline from the presence of blood. The specific gravity is low or high; it may be high from the presence of albumin. The normal solids are diminished both absolutely and relatively, especially the chlorine and urea. The albumin varies from one-fourth to one-half of one per cent. The sediment is abundant and is of a dark-brown color. There are large numbers of normal and decolorized blood-corpuscles and brown granular renal cells. Brown granular, epithelial, blood, fibrinous, and even hyaline and finely granular casts are seen in abundance. There may also be evidence of an *acute pyelitis*, as shown by clumps of caudate cells, round cells, and leucocytes. This stage lasts from five to ten days.

Second or Fatty Stage.—In this stage the quantity of urine and solids excreted begins to increase, the albumin to diminish, and the sediment to become more dilute, with the same characteristics, but with the addition of fatty renal cells, fatty casts, and compound granule-cells. This stage lasts about as long as the first stage.

Third Stage, or Stage of Convalescence.—In this stage there is a marked increase in the quantity of urine, which gradually loses its dark and smoky color and becomes pale, faintly acid, of low specific gravity, and with only a trace of albumin. The solids are nearly normal in absolute amount, but relatively are diminished, owing to the increased amount of urine. The sediment becomes slight in quantity, colorless, and shows a great diminution in the number of renal elements seen in the fatty stage. This stage may last from several months to several years, and be followed by complete recovery, especially in children.

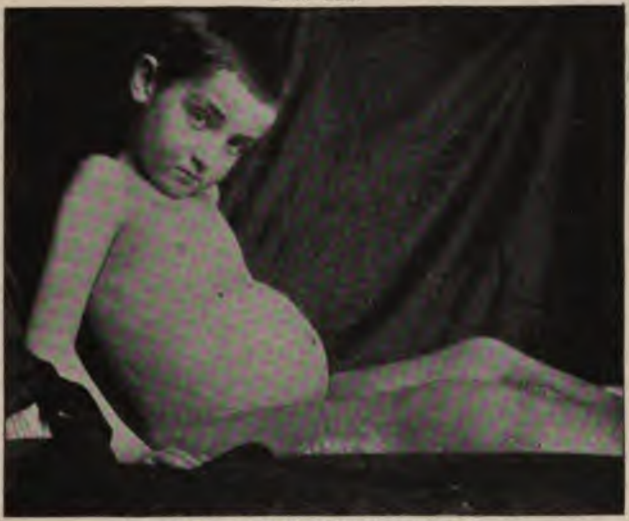
Exacerbations may occur at any time in the course of the second and third stages, and be followed by the return of the characteristics of the first stage. There is a sudden drop in the quantity of urine, with the reappearance or increase of the blood elements, especially of fresh red blood-corpuscles, the amount depending upon the intensity of the exacerbations.

COMPLICATIONS.—Pneumonia, pleurisy, endocarditis, and pericarditis should all be carefully watched for, as they may occur any time in the course of an acute nephritis. Uræmia, however, is the condition especially to be guarded against. With a fall in the amount of urine and diminution in the amount of solids, as judged by a daily examination of the urea, special measures should be taken to increase the elimination of the toxic substances by diuresis, diaphoresis, or free catharsis.

DIFFERENTIAL DIAGNOSIS.—The distinction between a severe acute hyperæmia and a mild acute nephritis is to be made by the sudden onset, œdema, and persistence of albumin, blood, and casts in considerable amounts.

In the convalescent stage of an acute nephritis in which there is a large amount of urine and a small percentage of albumin, the characteristics of the urine resemble those which occur in a chronic interstitial nephritis, but the history of an acute attack, the normal solids and presence of blood, and the rarity of chronic nephritis in children enable us to make the diagnosis if several examinations of the urine are made.

FIG. 182.



Acute diffuse nephritis, following scarlet fever.

Cardiac disease with dropsy is to be distinguished either from acute diffuse nephritis or subacute glomerular nephritis by the evidences of valvular lesions and the character of the urine in cardiac disease, which is that of a passive hyperæmia or congestion.

Fig. 182 illustrates the character of the dropsy which may occur in the course of an acute nephritis. The case is described on page 577.

TREATMENT.—The treatment of acute nephritis consists primarily in the administration of a strict milk diet, rest in bed, and the free use of diuretics. The details of the treatment and of the complications which may arise, such as uræmia, have been sufficiently described in connection with nephritis following scarlet fever on page 573.

of nephritis corresponds to the condition of urine seen in *active hyperæmia.* (Ogden.)

SYMPTOMS.—The symptoms of acute nephritis are such as have been already described under scarlet fever on page 572. In general, the symptoms arising in cases due to other causes than scarlet fever are the same, but less severe than those described in connection with that disease. Briefly they may be stated to be, sudden onset with mild fever; rapid development of anæmia; swelling of the eyelids, face, ankles, and often general anasarca; headache, thirst, nausea, and vomiting; frequent micturition, but diminished quantity; and in case of the advent of uræmia, stupor and convulsions.

Urine.—The characteristics of the urine are generally described according to three recognizable stages.

First or Acute Stage.—The quantity is very much diminished, at times almost suppressed. The color is dark, smoky, or blood-red. The reaction is acid, rarely alkaline from the presence of blood. The specific gravity is low or high; it may be high from the presence of albumin. The normal solids are diminished both absolutely and relatively, especially the chlorine and urea. The albumin varies from one-fourth to one-half of one per cent. The sediment is abundant and is of a dark-brown color. There are large numbers of normal and decolorized blood-corpuscles and brown granular renal cells. Brown granular, epithelial, blood, fibrinous, and even hyaline and finely granular casts are seen in abundance. There may also be evidence of an *acute pyelitis*, as shown by clumps of caudate cells, round cells, and leucocytes. This stage lasts from five to ten days.

Second or Fatty Stage.—In this stage the quantity of urine and solids excreted begins to increase, the albumin to diminish, and the sediment to become more dilute, with the same characteristics, but with the addition of fatty renal cells, fatty casts, and compound granule-cells. This stage lasts about as long as the first stage.

Third Stage, or Stage of Convalescence.—In this stage there is a marked increase in the quantity of urine, which gradually loses its dark and smoky color and becomes pale, faintly acid, of low specific gravity, and with only a trace of albumin. The solids are nearly normal in absolute amount, but relatively are diminished, owing to the increased amount of urine. The sediment becomes slight in quantity, colorless, and shows a great diminution in the number of renal elements seen in the fatty stage. This stage may last from several months to several years, and be followed by complete recovery, especially in children.

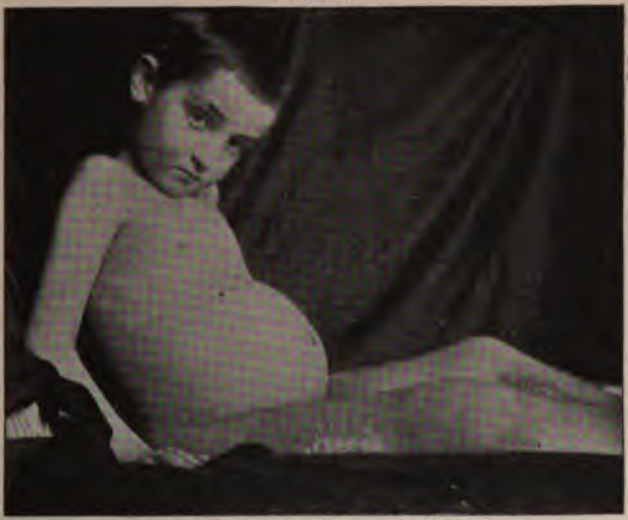
Exacerbations may occur at any time in the course of the second and third stages, and be followed by the return of the characteristics of the first stage. There is a sudden drop in the quantity of urine, with the reappearance or increase of the blood elements, especially of fresh red blood-corpuscles, the amount depending upon the intensity of the exacerbations.

Complications.—Pneumonia, pleurisy, endocarditis, and pericarditis should all be carefully watched for, as they may occur any time in the course of an acute nephritis. Uræmia, however, is the condition especially to be guarded against. With a fall in the amount of urine and diminution in the amount of solids, as judged by a daily examination of the urea, special measures should be taken to increase the elimination of the toxic substances by diuresis, diaphoresis, or free catharsis.

Differential Diagnosis.—The distinction between a severe acute hyperæmia and a mild acute nephritis is to be made by the sudden onset, œdema, and persistence of albumin, blood, and casts in considerable amounts.

In the convalescent stage of an acute nephritis in which there is a large amount of urine and a small percentage of albumin, the characteristics of the urine resemble those which occur in a chronic interstitial nephritis, but the history of an acute attack, the normal solids and presence of blood, and the rarity of chronic nephritis in children enable us to make the diagnosis if several examinations of the urine are made.

Fig. 182.



Acute diffuse nephritis, following scarlet fever.

Cardiac disease with dropsy is to be distinguished either from acute diffuse nephritis or subacute glomerular nephritis by the evidences of valvular lesions and the character of the urine in cardiac disease, which is that of a passive hyperæmia or congestion.

Fig. 182 illustrates the character of the dropsy which may occur in the course of an acute nephritis. The case is described on page 577.

Treatment.—The treatment of acute nephritis consists primarily in the administration of a strict milk diet, rest in bed, and the free use of diuretics. The details of the treatment and of the complications which may arise, such as uræmia, have been sufficiently described in connection with nephritis following scarlet fever on page 573.

SUBACUTE GLOMERULAR NEPHRITIS.

This condition is known also as "*chronic parenchymatous nephritis.*"

ETIOLOGY.—Chronic parenchymatous nephritis is not a common disease in childhood, and its etiology is still very obscure. Some cases have followed an attack of acute nephritis, and in these there has generally been an interval during which the urine has contained simply a trace of albumin and a few casts, the symptoms of a chronic affection of the kidney appearing later. Cases have also occurred in connection with long-continued suppurative processes in the bones, joints, or elsewhere, arising in the course of tuberculosis or syphilis. In these cases amyloid infiltration is also apt to occur. In the majority of instances no cause whatever can be discovered.

PATHOLOGY.—The kidney is enlarged, pale, slightly mottled, and of increased consistency. The cortex is increased in width and is pale. The markings are indistinct. The essential lesions consist of swelling and increase in the nuclear elements of the glomeruli, with hyaline degeneration of the blood-vessels. There is proliferation and desquamation of the capsular epithelium and new formation of connective tissue. Diffuse degeneration and desquamation of the tubular epithelium and œdema of the intertubular tissue occur. (Councilman.)

SYMPTOMS.—The onset of subacute glomerular nephritis, or chronic parenchymatous nephritis, is sometimes insidious. Indigestion, loss of appetite, and vomiting are early symptoms. The face becomes pallid and "pasty," and œdema of the face, eyes, and ankles develops. Palpitation, headache, and dyspnœa, with hypertrophy of the heart and pulse of increased tension, are present and are often early symptoms. Epistaxis may occur. Nervous symptoms of a uræmic type are often pronounced, and in the more advanced stages of the disease may end in convulsions and coma. Changes in the retina are often found, and there is always a special tendency towards the development of intercurrent diseases, such as pneumonia and pleurisy, which often lead to a fatal conclusion. As the disease advances the œdema becomes more general, and a condition of anasarca develops. The anæmia becomes more profound and the cardiac symptoms more marked.

Urine.—Micturition is frequent, but the quantity of urine which is passed is small. The disease is characterized by periods of activity and quiescence. In the active stages the œdema is increased, the quantity of urine is very small, and uræmic symptoms threaten. The urine is high-colored, strongly acid, with high specific gravity, and the solids are absolutely much diminished, although relatively they may be increased. The albumin is always very large, averaging from one-half to one per cent., although it is sometimes much higher. The sediment consists of many hyaline, granular, and fatty casts, some of which contain fatty renal cells and compound granule-cells adherent. These fatty degenerated cells are also found free. In advanced stages waxy casts will be met with and are usually of bad prognosis. Signs of acute irritation of the kidney are often present and are indicated by the presence of blood elements.

During the quiescent stage the dropsy becomes absorbed, and the œdema may entirely disappear. This change is associated with a marked increase in the quantity of urine, which may become normal or even in excess of the normal limit. As a result, the urine becomes pale, of a lower specific gravity, and with solids which are both relatively and absolutely diminished. The albumin remains high, ranging from one-fourth to one-half per cent. The sediment is the same as in the active stage, but the renal elements are less numerous.

Diagnosis.—It is important not to diagnosticate this form of parenchymatous nephritis when complicated with an active hyperæmia with the second stage of an acute nephritis, for the prognosis is very different in the two conditions. The diagnosis can only be made by attention to the history of the case and to the changes which take place in the urine. If the case is one of a subacute glomerular nephritis complicated with hyperæmia, the blood elements of the acute process will subside in the course of a few weeks.

Prognosis.—The prognosis is very grave. A few very rare cases of recovery have been reported. Most cases, however, die from uræmic intoxication or from some intercurrent disease, such as pneumonia. There may be a remission in the symptoms for a time. The duration of the disease is from one to five years.

Treatment.—The treatment of chronic parenchymatous nephritis is essentially the same as in acute glomerular nephritis. The diet should be restricted as far as possible to milk. Good hygienic surroundings and as much rest as possible are indicated.

The use of diuretics and the treatment of general œdema and threatened uræmia have been described in detail in the chapter on scarlet fever (page 573). The anæmia is to be treated with iron, especially the tincture of the chloride, but the indications for iron should be determined by the blood examination and not from the pallor. Exposure to cold must be avoided, and residence in a warm, even climate during the winter months is very desirable. The skin must be kept active by bathing and friction, and moderate, regular exercise taken when the child is strong enough. Cardiac dilatation is to be combated with digitalis and strychnine.

CHRONIC INTERSTITIAL NEPHRITIS.

Chronic interstitial nephritis (*chronic Bright's disease*) is very uncommon in childhood. It is hardly practicable to attempt to make the distinction between the various forms of chronic nephritis as can often be done in the case of adults. I therefore shall not describe chronic interstitial nephritis, chronic diffuse nephritis of the interstitial type, and chronic diffuse nephritis of the parenchymatous type as separate diseases.

Etiology.—The etiology of chronic interstitial nephritis is obscure. Hereditary tendencies and congenital syphilis play a part in the production of the disease in young children, but the common predisposing or associated conditions which are found in adults, such as gout, lead poisoning, chronic alcoholism, and arterio-sclerosis are very rare in childhood. In some cases a subacute glomerular or chronic parenchymatous nephritis is followed by a chronic interstitial nephritis.

Pathology.—The pathological conditions do not differ from the lesions as seen in the adult. The kidneys are very small and contracted with thick adherent capsules. The color is reddish, and on section the cortex is seen to be thin, the pyramids wasted, and the arteries thickened. The glomeruli are injected, and the pyramids show passive congestion. On microscopic examination the connective tissue between the tubules is much increased. The epithelium is degenerated. A general condition of arterio-sclerosis prevails.

Symptoms and Diagnosis.—The diagnosis can scarcely be made from the symptoms. The disease is progressive and slow, with no characteristic symptoms. Cases have been reported in which there were headache, weakness, dyspnœa, palpitation, and disturbance of vision. Hypertrophy of the left ventricle, with a pulse of increased tension, occurs as a constant lesion. There is little tendency to œdema; retinitis may be present. Sometimes severe nervous symptoms are prominent, such as tremor, increased reflexes, aphasia, and psychoses. Cerebral hemorrhages may occur as in adults. Baginsky refers to the lack of development of the children in these cases, and this condition was noticed in a case of this disease which occurred at the Boston Children's Hospital.

This child, a girl, twelve years old, showed the development of a child of about seven years. The only symptom until she died of uræmic poisoning was persistent headache. The post-mortem examination showed marked interstitial nephritis, but it was not possible to determine whether it was primary or not, and no previous history could be obtained.

Urine.—In chronic interstitial nephritis the amount of urine passed in the twenty-four hours is very greatly increased. It has a low specific gravity, a very slight sediment, and a trace of albumin. The microscopic examination shows a few hyaline and finely granular casts and occasional renal cells. Sometimes towards the end of the disease highly refractive homogeneous casts resembling wax appear in the urine. At this time the amount of urine may be somewhat diminished, but the specific gravity does not rise, as the excretion of urea is interfered with.

Diagnosis.—The early stages are not recognizable. The cases demand careful observation for a long time and repeated examinations of the urine. The presence of cardio-vascular changes, such as cardiac enlargement and a pulse of high tension, are important. The condition must be distinguished from physiological albuminuria and from passive hyperæmia.

The distinction must be made principally on the clinical evidence of the symptoms and of the physical examination.

PROGNOSIS.—The prognosis is very unfavorable. The children usually die of cerebral hemorrhage or of some intercurrent disease. It is impossible to predict the duration of the disease from the symptoms or from the urinary examination. The presence of marked cardio-vascular changes, of œdema from failing compensation of the hypertrophied heart, and the continued excretion of very small amounts of solids, especially of urea, are unfavorable signs.

TREATMENT.—The treatment of chronic interstitial nephritis is essentially the same as that of subacute glomerular nephritis. The diet, however, need not be so strictly limited. It should be light and nutritious and moderate in amount. Meat may be given in small quantities. Alcohol should be prohibited. Free use of the mineral waters is beneficial. In general, the indications are to maintain the functional activity of the heart, kidneys, skin, and bowels according to the methods which have been described.

When the arterial tension is high, nitroglycerin may be tried. As evidences of failing compensation of the heart develop, appropriate supportive treatment with strychnine and digitalis should be given. Threatened uræmia is to be met by the same measures which have been described in connection with acute nephritis (page 573).

AMYLOID INFILTRATION.

Amyloid infiltration of the kidney may occur in the kidney in connection with amyloid changes in other organs, especially in the liver, spleen, and intestines. It is always combined with other changes in the kidney, usually those of a chronic parenchymatous nephritis, and it is not to be considered as a separate disease of the kidney.

ETIOLOGY.—It occurs at times in connection with chronic suppurative processes in the bones or elsewhere, and also in tuberculosis, syphilis, and chronic wasting diseases. It is not, however, especially common in early life.

PATHOLOGY.—The amyloid change usually begins in the vessels of the glomerulus of the kidney, extends to other small vessels, and finally to the walls and epithelium of the tubules. The kidneys always show signs of diffuse nephritis.

SYMPTOMS.—The symptoms are those of chronic nephritis. The character of the urine, the presence of a cause, and of amyloid changes in the liver and spleen, shown clinically by enlargement, are the signs by which the diagnosis is made. Hypertrophy of the heart is rare. The urine is usually passed in large quantity when the amyloid changes are advanced. The specific gravity is low, and albumin is present to the extent of from one-tenth of one per cent. to one per cent. When the amount of urine is not much increased, as may happen temporarily, the albumin occurs in large amount. Microscopic examination shows no characteristic sediment; but when, as may often happen, the disease is combined with chronic nephritis, the sediment will show evidence of this latter disease. Waxy casts appear much earlier than in other forms of chronic nephritis. There are no fatty elements unless the disease is complicated.

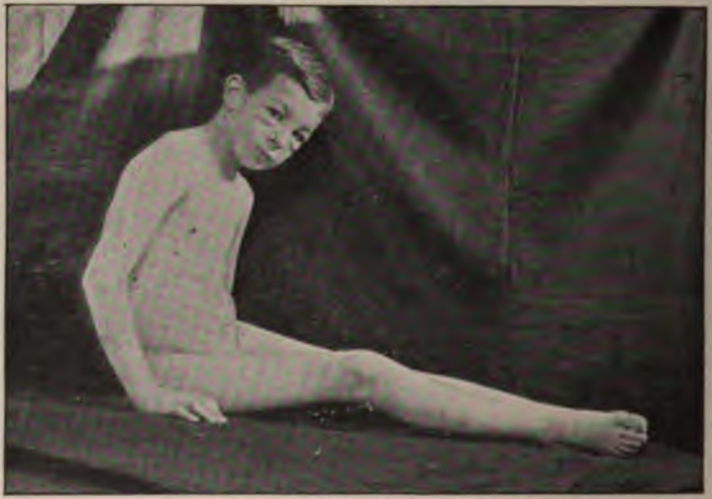
PROGNOSIS.—On account of the usual causes of this condition the prognosis is unfavorable.

TREATMENT.—The treatment is the same as that of chronic interstitial nephritis and of the primary disease upon which the amyloid changes are dependent.

The case which follows illustrates a chronic parenchymatous nephritis with an acute exacerbation.

The boy was eleven years old, and had had nephritis for one year. He had pertussis when he was three years old, scarlet fever when he was four years old, and measles and pneumonia when he was five years old. He is reported to have remained well from that time until nine months before he came under my observation, when, without any known cause, such as exposure to cold or sickness of any kind, his face and eyes began to be œdematous. This was followed by œdema of the legs and ankles, and was accompanied by dyspnœa. The urine was noticed to be nearly of the color of blood, and to be lessened in amount. He was kept in bed for six weeks, and is said not to have complained of any especial discomfort. During this attack his appetite remained fair. From the beginning of the attack he grew weak and pale. Six weeks before the present history the paleness and œdema about the eyes increased, and the urine became smoky again. This was followed by œdema of the ankles, feet, and legs, accompanied by dyspnœa. The bowels were regular, and there was no vomiting. Sleep was not disturbed. On entering the hospital his face looked pale and waxy. There was considerable œdema of the face, especially of the eyes. His tongue was slightly coated, and there was œdema of the ankles, feet, and legs.

FIG. 183.



Probable chronic parenchymatous nephritis with an acute exacerbation. Male, 11 years old. Relapse after being out of bed five days.

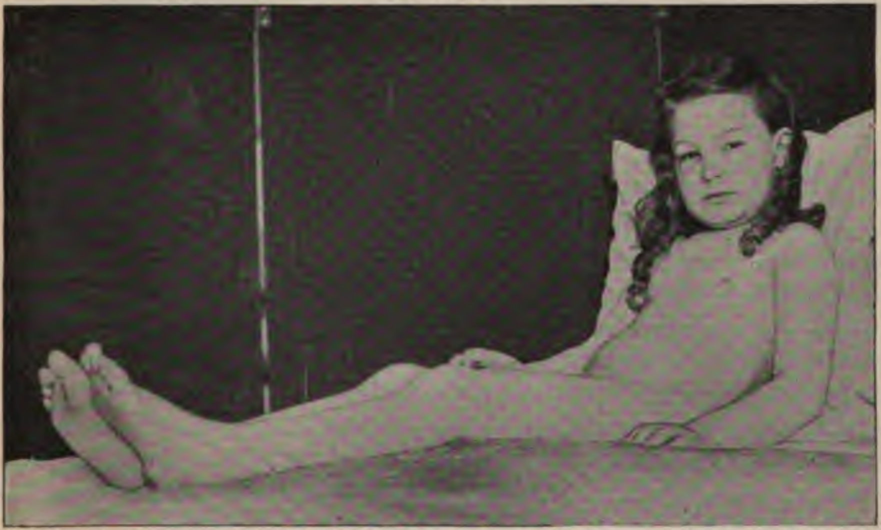
Nothing abnormal was found in the heart or lungs, and there was no evidence of ascites.

He was kept in bed and given a diet of milk. Under this treatment the œdema and anæmia disappeared rapidly, and in two weeks he was allowed to be dressed and about the ward. Five days later he again had œdema of the face, and was immediately put to bed. From 750 to 900 c.c. (25 to 30 ounces) of urine were passed in the twenty-four hours. An examination showed it to have a specific gravity of 1010, an acid reaction, to contain about 9 grammes (140 grains) of urea in the twenty-four-hour quantity, to have the chlorides diminished, and to contain 0.6 of one per cent. of albumin, but no sugar. The sediment showed numerous hyaline casts of medium diameter, some of large diameter from the straight tubules, many coarse and fine granular casts, numerous fibrinous casts, and many casts with renal cells adherent; also epithelial casts and blood casts; an excess of renal epithelium, most of it granular or fatty; a large amount of abnormal blood, free fat, and fatty casts. His temperature varied from 36.6° to 37.2° C. (98° to 99° F.).

After remaining in the hospital for two months, with temporary periods of improvement, he was discharged in about the same condition as when he entered.

The following represents another case of the same type.

FIG. 184.



Probable chronic parenchymatous nephritis with an acute exacerbation. Female, 9 years old. Second week of the disease.

This child had measles when she was two years old, scarlet fever when she was three years old, varicella when she was six years old, and pertussis when she was eight years old. She apparently recovered entirely from all these diseases, and was well until one week before entering the hospital, when, without any apparent cause, her face and feet began to swell. She complained of no pain, and had no other symptoms. There was marked and extensive œdema of the entire face, body, and limbs. There was also a pronounced pallor of the skin. Nothing abnormal was detected in the heart or lungs. There was no ascites, headache, nor discomfort.

An examination of the urine showed the color to be pale, the reaction acid, the specific gravity 1012, and the sediment moderate; it contained 0.25+ of one per cent. albumin, and no sugar; the sediment contained considerable abnormal blood, some free fat, and a number of hyaline and fine granular casts of medium and small diameter, many of them short and with fat-globules adherent. There were some fatty renal epithelium, leucocytes, casts with renal epithelium, and hyaline casts with a few renal cells adherent. There were also several fatty casts. The casts were not very numerous.

She was treated by absolute rest in bed, bitartrate of potassium, digitalis, and a diet of milk.

In about a week the œdema rapidly diminished and the urine increased in amount. An analysis of the urine at this time showed that the color was pale, that it had a specific gravity of 1010, a trace of albumin, and a slight sediment, consisting of a small amount of blood, renal epithelium, and a few casts with blood. The total amount of urine passed in the twenty-four hours was 2010 c.c. (67 ounces).

An examination of the urine three weeks later showed the color to be pale, the reaction acid, the specific gravity 1014, the albumin 0.25+ of one per cent. It contained hyaline and fine granular casts of small diameter, many with fat-globules and renal cells adherent; also free fat-globules, fatty and granular renal epithelium, some normal and abnormal blood, leucocytes, and squamous cells. The casts were not very numerous, and there was not much change from what was found in the urine three weeks previously. At this time the urine again became scanty, and the œdema and pallor returned, but she did not complain of any discomfort. An examination of the urine eight weeks later showed it to be pale and cloudy, the reaction acid, the specific gravity 1018, and that it contained considerable sediment, and albumin 0.25+ of one per cent. The sediment consisted chiefly of hyaline casts of medium and small diameter, many of them having renal cells and fat adherent. There were also a few finely granular casts, considerable abnormal blood, free fat, fatty renal cells, epithelium, leucocytes, and occasionally blood, epithelial, and fatty casts.

ACUTE PYELITIS AND PYELONEPHRITIS.

ETIOLOGY.—Pyelitis is an inflammation of the mucous membrane lining the pelvis of the kidney. After the pyelitis has lasted for a time, the substance of the kidney may be involved and pyelonephritis results. The most common cause is the excretion of uric acid by the kidney, or pelvic calculi. It may also be due to an extension upward of gonorrhœa or cystitis from the ureter and bladder, or to tuberculosis of the kidney, or to malignant growths. There is also an infectious form which may appear in the acute infectious fevers, such as scarlet fever, typhoid, or malaria. Pyelitis due to a local cause is usually unilateral. The form which appears as a complication in fevers is usually bilateral. An acute pyelonephritis not infrequently follows exposure to cold or wet.

PATHOLOGY.—The acute cases show an acute catarrhal inflammation of the mucous membrane of the pelvis of the kidney. In chronic cases there is thickening of the membrane and occasionally a considerable accumulation of pus. In pyelonephritis the suppurative process extends into the kidney itself.

SYMPTOMS.—In an acute attack of the disease, such as is caused by uric acid or a calculus, there are often local pain, chills, and fever. Typical attacks of renal colic, with vomiting, pain, and intermittent fever, may occur. Hæmaturia may be an early symptom. Micturition is frequent. If the condition be due to tuberculosis, malignant growths, or abscess of the kidney, there will be more or less cachexia and emaciation, and there may be local pain and tenderness. Pyuria is often the only symptom in the cases complicating acute infectious diseases. Leucocytosis is generally present.

Urine.—The diagnosis is made from the examination of the urine, the characteristics of which vary according to the extent to which the inflammation involves the parenchyma of the kidney. The quantity of urine excreted is small. The color is high, smoky, or blood-red, according to the amount of blood present. The specific gravity is generally high and the total solids absolutely diminished, but relatively increased. The quantity of albumin varies from a trace to one-fourth or to one-half of one per cent., or even higher, depending upon the severity of the involvement of the kidney itself. The microscopic examination shows sometimes the whole field to be filled with pus-corpuscles, at other times the pus is in clumps; there are also present small round cells with single nuclei, from the pelvis or from the kidney, and more or less blood. The diagnostic cell of pyelitis is the " caudate cell," which is a small cell about the size of a renal cell, having a single nucleus and a tail. These cells come from the superficial layers of the pelvis of the kidney, and may disappear from the sediment as the inflammatory process becomes subacute or chronic. If the kidney is affected there are casts of various kinds, hyaline, granular, epithelial, and blood. The casts may be hard to find if the field is filled with pus. The presence of tubercle bacilli in the sediment, shown by appropriate methods of staining, establishes the diagnosis of tuberculosis. The tuberculin test may be used with advantage. When calculi are present, they usually cause local pain and tenderness and hæmaturia, and occasionally attacks of renal colic. In the freshly passed urine, uric acid is often present in the sediment in the form of irregular spiculated crystals.

DIAGNOSIS.—New growths are more apt to produce hemorrhages than pus. Pyelitis may usually be distinguished from cystitis by the history, acid reaction of the urine, the presence of caudate and round pelvic cells in large numbers, and the local signs. In cystitis, on the other hand, there is generally vesicle pain and tenesmus, an alkaline urine, and large numbers of squamous and prostatic cells. It may be confirmed by the aid of the cystoscope. Catheterization of the ureters in older children, when possible, is of great aid in making a definite diagnosis.

PROGNOSIS.—The prognosis depends upon the cause. In malignant growths it is fatal. This is true to a greater or less degree when the tubercle bacillus is the cause of the disease, as in almost every case tuberculosis is present somewhere else in the body. When uric acid or a calculus is the cause, the prognosis is more favorable under appropriate treatment.

TREATMENT.—Uric acid, if present, should be treated by neutralizing the acidity of the urine, by placing the child upon a mild and unirritating diet, such as milk, and by making it drink freely of water. Potassium citrate should be used to neutralize excessive acidity of the urine, or benzoic acid if the urine is alkaline. Either may be given in doses of from 0.12 to 0.30 gramme (2 to 5 grains) three or four times daily in from one to four ounces of water. Operative treatment is at times called for when a calculus is present.

CHRONIC PYELITIS.

ETIOLOGY.—Chronic pyelitis may occur from a number of causes, such as tuberculosis, as a result of acute pyelitis from any cause, from irritation of crystals and calculi, especially when the outflow of the urine through the ureter is obstructed, and from a movable kidney.

SYMPTOMS.—The symptoms depend largely upon the cause and the manner in which the disease develops. There are often no symptoms at all, and again we may have those of acute obstruction of the urine. If there is retention of pus, fever, sweats, leucocytosis, and renal colic result, and the condition is spoken of as *pyonephrosis*. If the pus is discharged into the bladder, these symptoms rapidly subside, returning if the retention recurs.

Urine.—The urine is somewhat diminished in amount, pale, and very turbid, slightly acid in reaction, of low specific gravity, and with the normal solids diminished both absolutely and relatively. The albumin varies from a slight trace to 0.1 per cent. The sediment is chiefly degenerated pus, with possibly a few blood-globules. Casts of large diameter from the straight tubules are generally present, but are hard to find. The presence of crystals may enable us to diagnosticate a pyelitis due to calculus, but concretions may exist without the crystals appearing in the urine. In all cases of chronic pyelitis the urine should be examined for tubercle bacilli.

PROGNOSIS.—The prognosis depends upon the cause. If the cause is removable and the involvement of the kidney is not severe, recovery may be expected.

TREATMENT.—The medical treatment is essentially the same as in acute pyelitis, but the case should be referred to the surgeon if there is a reasonable chance of removing the cause by operation.

PERINEPHRITIS.

Perinephritis is an inflammation in the connective tissue about the kidney. It may end in resolution or suppuration. It has been confounded not infrequently with hip disease or vertebral caries. The most important paper on perinephritis in children was published in 1880 by Gibney, who reported twenty-eight cases.

ETIOLOGY AND PATHOLOGY.—Perinephritis in children may be secondary to suppuration in the kidney, such as results from calculi or tuberculosis, or to disease of the suprarenal capsules or to trauma. It is more frequently primary. The cellular tissue about the kidney becomes inflamed, and in the majority of cases suppuration occurs. The pus may discharge outward in the ileo-costal space, or into the pleura or bowel.

SYMPTOMS.—The onset may be acute or chronic. In the acute cases there is high fever, often with a chill. There is usually gastric disturbance, and the constitutional symptoms may be very severe. The bowels are constipated at times. There is usually local pain referred either to the hip or small of the back. The spine is painful and rigid, and the leg on the same side is drawn up as the movement of extension is productive of pain. General movements of the body are instinctively avoided by the patient. There is tenderness and resistance in the lumbar region, and later a tumor appears, in which deep fluctuation may sometimes be obtained. The size of the abscess may be very large. The cases which discharge into the pleura are usually diagnosticated as empyema. The character of the urine is rarely affected. The condition runs its course in from a few weeks to six months.

DIAGNOSIS.—Hip disease is likely to be confused with perinephritis, but the diagnosis is easy after careful examination. In hip disease the process is much more chronic, and the deformity is produced insidiously. Moreover, the pain is lower down, and there is tenderness over the joints. There is limitation of all the movements of the hip instead of extension alone.

Perinephritis may give local signs similar to psoas abscess from spinal caries, but the characteristic changes and deformity of the lumbar vertebræ are absent.

Perinephritis may be confused with typhoid fever when the constitutional disturbance is severe and prolonged and the local signs develop slowly.

PROGNOSIS.—The primary cases almost always end in complete recovery. Peritonitis from rupture of the abscess is the most serious complication.

TREATMENT.—The treatment is surgical. Rest in bed and hot local applications are first indicated, and later incision with free drainage of the abscess.

HYDRONEPHROSIS.

Hydronephrosis is a condition in which the pelvis and calyces of the kidney are dilated with fluid as a result of an obstruction to the outflow of urine.

ETIOLOGY.—Hydronephrosis may be congenital, in which case it may be due to constriction of the ureter. Both kidneys may be affected, but usually only one is involved. When acquired it generally affects but one kidney, and may be caused by obstruction of urine either from above, such as may occur from an impacted calculus in the hilus of the kidney or in the urethra, or from below by the pressure from a tumor or from enlarged mesenteric glands. The effects are mechanical, and are due to the pressure on the kidney of the retained fluid, which leads to the gradual absorption of the parenchyma of the kidney. These tumors sometimes acquire a large size. They are frequently associated with other deformity or degeneration of the kidney.

Symptoms.—The main symptom of hydronephrosis is the presence of an abdominal tumor connected with the kidney. When the tumor has grown sufficiently large, fluctuation can usually be detected, and aspiration gives a fluid which ordinarily contains urea. Subjective symptoms are usually absent and urinary signs are rare. If only one kidney is affected, the other performs the function of both, and the general condition of the child may remain good. When both kidneys are affected, life is usually too short for the development of a tumor, and the condition is not recognized.

Prognosis.—Bilateral cases are usually fatal within a year. If only one kidney is involved operation gives the only hope of relief.

Treatment.—The treatment is surgical. Total excision of the kidney has given better results than incision and drainage.

MALIGNANT GROWTHS AND ENLARGEMENT.

Tumors of the kidney are more common in the child than in the adult. They are almost always primary and usually malignant. The simple adenomata are probably equally common in both, but the child is much more liable to carcinomata and sarcomata than is the adult. Sarcomata are the most common in the first five years of life, and usually occur in one kidney.

Symptoms and Diagnosis.—The diagnosis depends upon the recognition of a tumor of the kidney, the hæmaturia, and the progressive emaciation and cachexia which arise. At times there is pain, but, as a rule, pain is absent. The urine sometimes gives evidence of a pyelo-nephritis ; hæmaturia and albuminuria usually occur at intervals late in the disease, at a time when the tumor can be felt through the abdominal wall. The tumors are frequently of large size and rapid growth. They may cause symptoms by compression of the lungs, vena cava, or bowels. Ascites is sometimes present, and very rarely general peritonitis. Some of the characteristics of a tumor of the kidney are that it is located in the hypogastric and lumbar regions, is deep-seated, and is not so commonly to be felt in the umbilical region as are tumors of the retro-peritoneal glands. The tumor is irregularly rounded, and usually does not have a well-marked border, such as is found in enlargement of the spleen and liver. In these cases of sarcomata of the kidney the health at first is often not much affected, but there are progressive emaciation and enlargement of the abdomen, commonly without pain. Malignant growths of the kidney may be confused with hydronephrosis.

Prognosis.—The prognosis is very unfavorable, although temporary relief is sometimes obtained by means of surgical interference.

Treatment.—The treatment is essentially operative. The total mortality of the operation, including cases of recurrence with death, is over seventy-five per cent.

DISEASES OF THE BLADDER AND GENITALS.

ACUTE CYSTITIS.

Acute cystitis is not a common affection in infancy and childhood.

Etiology.—It may be caused by a vesical calculus, by irritants, such as turpentine, and also occasionally by the extension of infection through the genital tract. It is not an infrequent complication of typhoid fever from direct infection by the bacteria which are eliminated by the urine. Gonorrhœa and infections by the pyogenic organisms are direct causes of acute cystitis. It is more common in girls than in boys.

Symptoms.—The symptoms of acute cystitis in children do not differ from those which are met with in the adult. The chief symptom is frequent and painful micturition. This local symptom is usually accompanied by fever, which may be high, and by general symptoms of malaise, fretfulness, and crying from vesical pain and tenesmus. The urine is passed in small quantities, and, as a rule, is of a reddish color at first, and gradually becomes of a lighter color. The specific gravity is high. When freshly passed it is acid, but it quickly becomes alkaline; there is a heavy sediment, and it contains a trace of albumin. Microscopic examination shows chiefly pus in large quantities, squamous epithelium, and some blood. To establish the diagnosis in females it is necessary to obtain the urine by the catheter, or first to wash out the vagina thoroughly, as the epithelium of the vagina and that of the bladder are very similar.

Prognosis.—The prognosis of acute cystitis is good after the removal of the cause.

Treatment.—The especial cause of the attack must be looked for, and removed if possible. The child should be kept perfectly quiet in bed, and should be made to drink a great deal of water. The diet should be of milk. In the acute cystitis occurring in the course of typhoid fever, urotropin in doses of from 0.06 to 0.32 gramme (1 to 5 grains) four times a day acts almost as a specific in from four to seven days. In cystitis due to other causes it is not so serviceable, except as a preventive of fermentation of urine within the bladder. Sedatives should be used freely.

CHRONIC CYSTITIS.

Etiology.—Chronic cystitis may be caused in children, as in adults, by a vesical calculus, by foreign bodies in the bladder, by tumors, by papillomata, and by tuberculosis. The nuclei of the calculi are generally composed of uric acid, upon which phosphates are precipitated in alkaline urine, and this deposition is favored by the accompanying catarrhal inflammation.

Symptoms.—Micturition is frequent and at times painful. Later there may be a constant dribbling of urine, giving rise to an offensive ammoniacal odor and causing irritation about the genitals. When there is a calculus in the bladder the stream is often suddenly interrupted during micturition, and the pain is more severe. Prolapse of the rectum is not uncommon with stone. In addition to these local symptoms there are general symptoms of anæmia and loss of weight. The urine is ammoniacal, offensive in odor, and turbid, has a heavy ropy sediment, and contains a trace of albumin. The sediment should be examined as soon as possible after the urine is passed, because the ammonia which is produced from the urea disintegrates the cells. The examination will show a large quantity of pus, some blood, bladder-epithelium, and crystals of triple phosphate and urate of ammonium. Tubercle bacilli may be found in the urine in tuberculosis of the bladder, but a prolonged and careful search is often necessary.

PROGNOSIS.—The prognosis of chronic cystitis depends upon the cause, upon the length of time during which the disease has persisted, and the presence or absence of a secondary infection of the kidney.

TREATMENT.—The urine should be diluted by giving distilled water in large amount. It may be rendered less irritating by such drugs as salol and buchu, and less alkaline by benzoate of sodium. Urotropin given by the mouth has yielded excellent results by disinfecting the urine in the bladder and checking alkaline fermentation. Washing out the bladder with weak solutions of permanganate of potash, creolin, boracic acid, or lysol is of use in many cases, and local applications may be made in tuberculosis of the organ. Operative treatment is indicated when a calculus is causing the disturbance.

At times it is exceedingly difficult to determine by the general symptoms whether or not a calculus is present in the bladder. The following case may be cited as an illustration :

A boy, seven years old, began to have pain of a spasmodic character in the region of the bladder during micturition. In connection with the pain there was a sudden stoppage of the flow of the urine and a bearing-down feeling in the rectum. These symptoms simulated those of a vesicle calculus so closely as to render a differential diagnosis very difficult. The boy was of a nervous temperament, and was rather anæmic, but otherwise was well and strong. Nothing abnormal was detected about the prepuce or the rectum. The pain was so annoying and caused so much trouble that it was deemed advisable to have the bladder examined for stone. An examination was made, but nothing abnormal was detected. After this a decided improvement took place, apparently connected with the passing of the sound, and the boy recovered entirely after remaining at home from school for a few weeks and having daily exercise in the open air.

VULVO-VAGINITIS.

Vulvo-vaginitis is a very common affection in little girls. It arises from a variety of irritations, one of which is the oxyuris vermicularis. In a very large number of cases the gonococcus of Neisser has been found in the purulent secretion. The gonococcus was found in all of six cases lately treated at the Boston Children's Hospital. The disease may also arise in children who are very much debilitated, and is met with at times in scarlet fever and in measles. Again, it is not infrequent in anæmic girls, in whom it occurs without any apparent cause.

PATHOLOGY.—The labia are reddened and are more or less swollen. There is a thick, purulent discharge of a greenish-yellow color, usually offensive. At times there is more or less excoriation of the inner surfaces of the labia. The inguinal glands may be slightly enlarged and tender. The urethra is, as a rule, involved in the irritation, and is swollen and red.

SYMPTOMS.—There may be some fever in the early stages of vulvo-vaginitis. Smarting and burning are usually complained of, but at times the staining of the clothing first calls attention to the disease. The children commonly become pale if the disease persists for some time. Micturition is painful and frequent in some cases, and the disease is one of the many causes of dysuria. In many cases the children appear to be quite well, with the exception of the local condition. A positive diagnosis of gonorrhœa is only possible by means of a microscopic examination of the discharge after staining by Gram's method.

PROGNOSIS.—The prognosis is good, but the disease is apt to be prolonged for several weeks or months. Complications may arise from the infection of the bladder, uterus, Fallopian tubes, inguinal glands, joints, or conjunctivæ. Infection of the conjunctivæ and uterus is common, infection of the other organs is much less so.

TREATMENT.—Local applications to the vagina constitute the only satisfactory form of treatment. This is difficult in young children, but may be accomplished with a soft rubber catheter. Such solutions as boracic acid 1 to 40, corrosive sublimate 1 to 5000, or creolin 1 to 500, may be used. In some severe cases local applications of nitrate of silver 1 or 2 per cent., or protargol, may be necessary. The labia should be kept separated by absorbent cotton, and the parts kept dry and covered with some mild dusting-powder. Absolute cleanliness must be observed, to prevent infection of the eyes and of other persons. The parts should be protected with compresses held in place by a bandage, which should be worn all the time, and the compresses should be frequently changed and burned. The towels used for the patient should not be left lying about, and should be carefully disinfected. Tonic treatment is sometimes indicated. The urine should be kept dilute, in order to avoid irritating the inflamed surfaces, and any complicating cystitis should be treated. During the active stage of the disease the child should be kept as quiet as possible, and on a diet of milk.

When the vulvo-vaginitis is caused by the oxyuris vermicularis, especial care should be given to eradicating the parasite from the rectum, as described on page 830. After this has been done, the vagina is readily freed from the parasite by using an injection of warm sweet oil, which is to be allowed to remain for three or four minutes, the vagina then being syringed out with warm water.

ORCHITIS.

Orchitis, or inflammation of the testis proper, occasionally results from direct injury, much more rarely from infection in gonorrhœa, syphilis, and tuberculosis. When present it is commonly accompanied by hydrocele. The orchitis which so commonly follows mumps in the adult is much less common in children. The treatment consists in support of the testicle by means of a suspensory or of a bandage.

EPIDIDYMITIS.

Acute epididymitis may be caused by trauma or by any irritation of the mucous membrane of the urethra. In this disease the whole scrotum is apt to be hot and tender, and the child is in great pain. The epididymis is much enlarged and exquisitely tender, and pushes the testis forward. The cord is often implicated, becoming enlarged and painful on pressure.

The treatment should be energetic, as, owing to the swelling of the tissues about the testicle, there may be so much pressure that the gland will be seriously damaged, although the subsequent atrophy may not declare itself for a considerable time. The child should be kept upon his back in bed, the bowels freed with a cathartic, and a series of hot poultices kept upon the scrotum. In all inflammations of the testis or epididymis the scrotum should be placed in such a position that the lower end of the testicle points upward.

TUMORS OF THE TESTICLE.

In addition to tubercular disease of the testis, tumors may be found in infancy and in early childhood. These may be congenital or acquired. The congenital tumors are very rare, and are usually of the dermoid variety. The most common of the acquired tumors are carcinomata and sarcomata, which are very malignant. The rapid growth and the large size of this variety usually render the diagnosis easy.

PHIMOSIS.

In early life there appears to be a physiological adhesion of the prepuce to the glans penis. As the child grows older these adhesions normally disappear. When the adhesion between the prepuce and the glans remains permanent and the prepuce is very tight, the condition gives rise to various symptoms. Thus the escape of the urine may be mechanically hindered, and the urine collecting behind the glans may give rise to irritation. Smegma is also apt to collect around the corona. In this way an inflammatory condition of the prepuce (posthitis) or of the glans (balanitis) may arise. As a result of this there is swelling, and micturition is painful and difficult. In addition to these local symptoms many secondary disturbances arise from the local reflex irritation. Among these are nervous phenomena of greater or less degree, such as insomnia or convulsions. Phimosis may lead to enuresis and masturbation.

In all cases of phimosis local treatment is indicated, and may be by dilatation, incision, or circumcision,—the latter being the most radical and producing the best results for complete relief from the morbid condition. In all cases, even if the phimosis is very slight, mechanical interference should be persisted in until absolute cleanliness can be secured, for in this way only will entire relief from the local and reflex symptoms be obtained.

ENURESIS.

Enuresis (*incontinence of urine*) is a condition in which there is an involuntary discharge of the urine. It may be continuous or periodic. It may also be diurnal, nocturnal, or both. It is of very frequent occurrence in infancy and early childhood. It is a symptom rather than a disease, and in most cases is a true neurosis. During the first year of life the infant has not learned to assume control of the mechanism of micturition, but during the second year this control is usually attained at an earlier or a later period according to the individual.

ETIOLOGY.—The causes of enuresis may be organic or functional, the latter in all probability being very commonly of a reflex nature.

Organic Enuresis.—The organic causes comprise such malformations as small ureters, a small bladder, exstrophy of the bladder, and hypospadias. Enuresis may also be caused by central lesions of the brain and cord, such as brain tumor, idiocy, or injury to the cord. The prognosis and treatment of these organic cases of enuresis vary according to the conditions which cause them, and need not be considered here.

Functional Enuresis.—In a large number of cases the children are of a highly nervous temperament, but enuresis is also often present in children who otherwise do not show any nervous symptoms. As has been stated by Rachford in an admirable paper on this subject, this condition may depend upon (1) irritable and unstable nerve-centres, (2) anæmia with malnutrition, and (3) reflex stimulation of certain nerve-centres in the lumbar cord. The longitudinal and circular muscular fibres of the bladder, which by their contraction empty the bladder, are innervated by sensory and motor nerves from the lumbar region of the cord, and the external sphincter in the prostatic portion of the urethra, which by its contraction prevents the escape of urine from the bladder, is also innervated by sensory and motor nerves from the lumbar cord. The researches of Von Zeissl show the manner in which reflex causes may act in starting or checking the flow of the urine. Thus, a reflex carried to the proper centre in the lumbar cord would, through the motor fibres of the erector nerve, contract the muscular coat of the bladder, and through the inhibitory fibres of the same nerve relax the sphincter vesicæ. In this manner the urine which is being expelled by the contracting bladder is allowed to pass without hinderance through the relaxed sphincter vesicæ. It is also to be remembered that the act of urination is in part under the control of the will. Admitting these anatomical and psychical facts, it is easily understood how the causes which produce enuresis may act in two ways: either directly on the centres in the lumbar cord, making them more irritable or unstable, and in that way increasing their reflex excitability, or indirectly through exaggerated reflex causes that affect both acceleratory and inhibitory influences sent to the bladder. These influences may be psychic, originating in the brain, or may be the result of external irritation originating in or near the bladder itself.

There is also during childhood a lack of development of the centres of inhibitory reflex acts, and in this way the muscular fibres of the bladder, having no inhibitory restraint, are excited to action by even so slight a reflex cause as a small quantity of urine in the bladder. For this reason enuresis is a normal condition during infancy, and ceases when the child's inhibitory mechanism is more developed (Soltmann). The inhibitory influence of the will is in abeyance during deep slumber, and nocturnal incontinence is therefore more frequent than diurnal. In any diseases which are accompanied by anæmia and malnutrition the reflex irritability of the lumbar nerve-centres is much increased and enuresis may result. Reflex enuresis may be caused by irritation in any portion of the genito-urinary tract, as by a vesical calculus, cystitis, vulvitis, phimosis, very acid urine, and over-filling of the bladder, as in diabetes, or by an irritation of some neighboring part, such as may arise from a fissure, polypus, or the oxyuris vermicularis in the rectum.

Symptoms.—As a symptom, enuresis is simply the involuntary emptying of the bladder.

Prognosis.—The prognosis of enuresis varies greatly, according to the cause and the individual. It is usually hopeless when it depends on disease of the brain or cord. In a large number of cases the enuresis lasts for only a short time, but in some cases it may continue throughout childhood; almost invariably, however, it ceases between the twelfth and the fourteenth year. The cases in which enuresis does not disappear at puberty are nearly always in girls.

Treatment.—The treatment of this functional form of enuresis is often prolonged and very unsatisfactory. According to my experience, in quite a number of cases the disease is intractable and is not affected by any treatment whatever, the individual finally recovering without treatment. Sometimes a habit of incontinence seems to have formed which continues after the removal of the original exciting cause. After a careful examination has shown that no malformation or central nervous lesion is present, the urine should be examined, to determine if it is abnormally acid. When this is found to be the cause of the irritation, a rapid cure can be effected in some cases by simply diluting the urine. In females, especially when there is irritation around the meatus urinarius, local applications are of great service, and in some cases dilatation of the urethra will produce a permanent cure. When phimosis is present, relief has sometimes been obtained by circumcision. The bowels should be regulated, little or no fluid should be given after five P.M., and it is well to have the child pass its water just before going to sleep, and to rouse it in the middle of the night in order that it may empty its bladder. The foot of the bed should be raised, in order that the urine shall not irritate the neck of the bladder. There is no especial drug which in my experience can be relied upon in curing enuresis. Attention to general hygiene is important. Plenty of sleep and a simple diet should be insisted upon. Punishment of the child is usually harmful, and should never be allowed. When the children are anæmic and debilitated, iron and nux vomica are indicated. When there is excessive irritability of the nerve-centres, belladonna and atropine are at times efficient in relieving this condition; but in many cases they fail to produce beneficial results even when given in toxic doses. Faradism applied to the perineum or to the base of the sacrum and to the symphysis pubis is in some cases beneficial. If the bladder is contracted, daily dilatation up to the normal size with warm water may be very effectual. There is, however, no routine treatment for enuresis. Each case should be studied closely, and in many instances when the especial cause of the condition has been found the enuresis can be relieved.

MASTURBATION.

Masturbation is one of the injurious habits of early life. It is most frequently practised during the later periods of childhood, especially between the tenth and fifteenth years. It may, however, become a habit at any age, even in infants of one year.

The inciting cause may be some local irritation, such as intestinal parasites, concentrated and very acid urine, phimosis, balanitis, vaginitis, and a long prepuce. It may also be inculcated by the indiscreet handling of the genitals by nurse-maids. Not infrequently the habit is started from imitation of other children.

There are no characteristic symptoms by which one can judge as to whether a child is a victim of this habit. The pallor, anæmia, dark rings under the eyes, headaches, and embarrassment which are sometimes mentioned as distinctive signs, are common to so many other conditions that they are not sufficient evidence on which to make a diagnosis. Masturbation is stated by some writers to be a direct cause of insanity.

The diagnosis of masturbation should only be made when the child has actually been observed to practise the habit on several occasions.

The treatment in young infants consists in mechanical restraint, the hands and legs being so tied that the act becomes impossible. In older children strict surveillance should be kept. Punishment should be avoided. Moral persuasion, diversion of the mind, exercise, bathing with cold water, diet, and general hygiene are the main points in the treatment. In serious cases the bromides may be given. Hypnotism has proved very beneficial in many of these cases.

DIVISION XV.

THE BLOOD. THE LYMPH-NODES. THE DUCTLESS GLANDS.

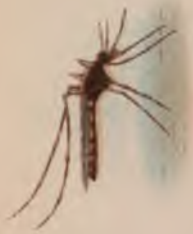
THE BLOOD IN INFANCY AND CHILDHOOD.

THE blood does not merely absorb the waste material from the tissues and carry fresh oxygenated material to replace it; it plays a far greater part than this in the economy, and is intimately connected with many diseases, both through its corpuscular elements and its serum. An immense amount of labor has been expended on examinations of the blood, both chemical and microscopical, in adults, but our knowledge of the blood as it occurs in pathological conditions in infancy and in early life is still very incomplete and is wanting in exactness.

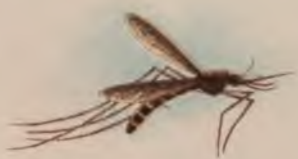
NOMENCLATURE.—The following nomenclature, with its accompanying plate, will explain the terms which are used in speaking of the various elements of the blood:

RED CORPUSCLES (Erythrocytes.)	Normal red corpuscles	(Plate XII.)
1. Hæmatoblasts	Nucleated red corpuscles	(Plate XII.)
(1) Normoblasts	Size of erythrocytes, having a small deeply staining nucleus	(Plate XII.)
(2) Megaloblasts	Large nucleated red corpuscles, having a large, often fragmented, nucleus, staining faintly	(Plate XII.)
(3) Microblasts	Small nucleated red corpuscles.	
2. Microcytes	Abnormally small erythrocytes	(Plate XII.)
3. Macrocytes or Megalocytes.	Abnormally large erythrocytes.	
4. Poikilocytes	Abnormally shaped erythrocytes	(Plate XII.)
WHITE CORPUSCLES	(Leucocytes.)	
1. Lymphocytes or small mononuclear. (Basophiles.)	Round mononuclear cells about the size of erythrocytes, with faintly staining protoplasm. The nucleus stains deeply, and fills nearly the whole cell	(Plate XII.)
2. Large Mononuclear and Transitional (Basophiles.)	Cells double the diameter of erythrocytes with oval, round, or indented (transitional), faintly staining nucleus filling a relatively small part of the cell	(Plate XII.)

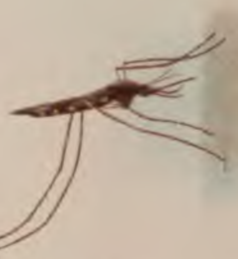
PLATE XII.



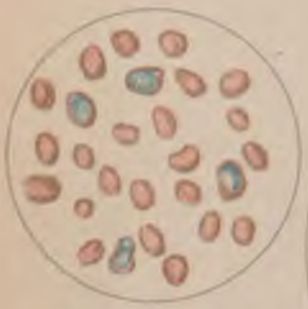
Culex.
(Resting Position.)



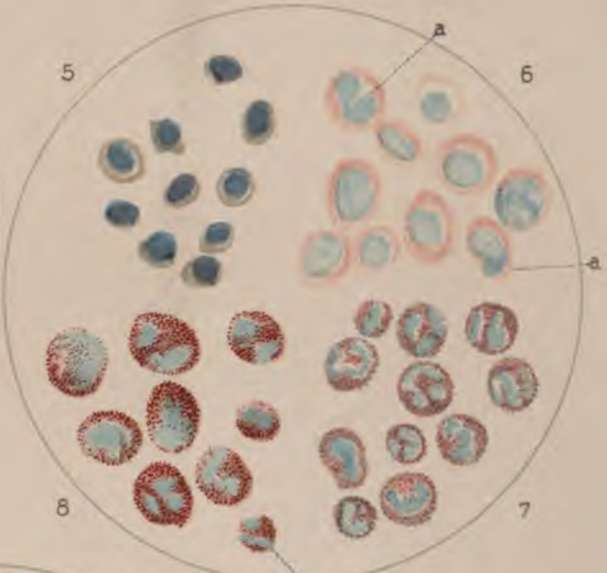
Anopheles.

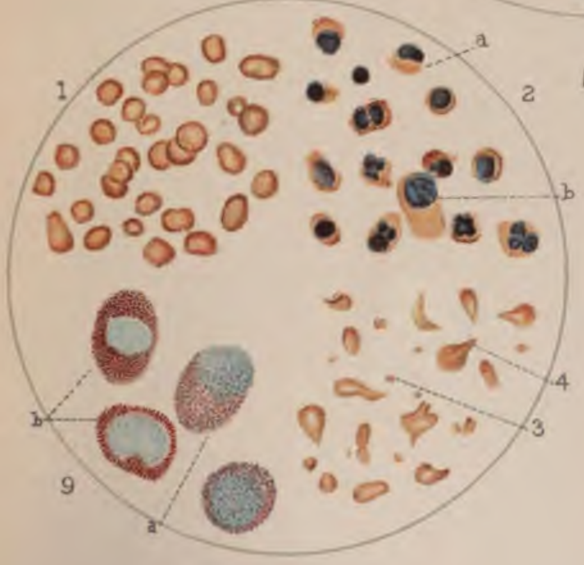


Anopheles.
(Resting Position.)



Plasmoidium Malariae
Oil Immers. REICHERT 1/15
Ocular No. 3.





LEITZ Oil Immers. 1/12 Ocular No. 3.

1. Normal Red Corpuscles.
2. Haematoblasts or Nucleated Red Corpuscles:
   a. Normoblasts.
   b. Megaloblasts.
3. Microcytes.
4. Poikilocytes.
5. Lymphocytes or Small Mononuclear.
6. Large Mononuclear:
   a. Transitional.
7. Polynuclear Neutrophiles.
8. Polynuclear Eosinophiles:
   a. Dwarf Eosinophile.
9. Myelocytes:
   a. Neutrophilic.
   b. Eosinophilic.

3. Neutrophiles or polynuclear cells.	Considered by most observers the oldest variety of the leucocytes. The nucleus stains with basic stains; the plasma stains faintly with neutral aniline stains, and the granules stain with a combination of both basic and acid stains, and hence are called neutrophiles. The nucleus is really polymorphous, though sometimes (apparently) broken	(Plate XII.)
4. Eosinophiles (Oxyphiles.)	Polymorphonuclear cells characterized by the presence of large, highly refractile granules which stain with all acid coloring matters	(Plate XII.)
5. Myelocytes or large mononuclear neutrophiles.	Large, round, or ovoid cells with one (seldom two) large faintly staining nuclei. The plasma is filled with small granules that take (*a*) a neutral or (*b*) an acid stain	(Plate XII.)
(*a*) Neutrophilic		(Plate XII.)
(*b*) Eosinophilic		(Plate XII.)
LEUCOCYTOSIS	An increase in the whole number of leucocytes, or an increase in the proportion of any single variety.	
BASOPHILIC	Stained by basic stains.	
ACIDOPHILIC or Eosinophilic.	Stained by acid stains.	
NEUTROPHILIC	Stained by neutral stains.	
POLYCHROMATOPHILIC	Taking more than one stain.	
OLIGOCYTHÆMIA	Reduction in number of erythrocytes.	
HÆMOLYSIS	Destruction of erythrocytes.	
HÆMOGLOBIN	Coloring matter of the blood.	
HÆMOGLOBINÆMIA	Presence of hæmoglobin in the serum.	
HÆMOGLOBINURIA	Presence of hæmoglobin in the urine.	

Plate XII., facing page 874, shows all the principal normal and abnormal cells occurring in the blood in early life. The different types of anæmia are characterized by a combination of the different cells in varying proportions, and by becoming familiar with their microscopic appearance, and then in a given case by calculating the percentages of the different cells which are present we have a very valuable aid in diagnosis.

THE BLOOD IN NORMAL CONDITIONS.

There are certain general characteristics which must be recognized when we are dealing with human beings in the earlier stages of their development.

These characteristics merely impress upon us that the blood, like the various organs and tissues, changes by a process of development from the fœtal to the developed condition of adult life. It is therefore necessary to take into consideration the age and stage of development of the individual whose blood we are examining before we can state that such a blood is normal or abnormal. For instance, just as it would be abnormal for the anterior fontanelle to be closed at the sixth month and unclosed at the twentieth month, so it would be abnormal for the blood of an infant in the early weeks of life to show the same ratio of leucocytes to erythrocytes as it would in adult life, or for the leucocytes to show the percentage of lymphocytes and polynuclear neutrophiles in the same proportion as at a later period. In fact, what I have already stated concerning the different stages of physical development holds true in the case of the blood, where what can be considered normal and physiological at one period becomes, if it lasts into another period, abnormal and significant of disease. In order to understand, therefore, the diseased conditions of the blood we must remember what is normal at each period. We will next consider the normal condition of the blood at birth and its various stages of development.

Amount of Blood at Birth.—Welcker states that the total amount of blood at birth is one-nineteenth of the body-weight. His opinion is based on the examination of a poorly developed infant, in which the umbilical cord was ligatured immediately at birth.

Schuecking places the amount at one-fifteenth of the body-weight, from an examination of five full-term infants, without expressing the blood from the placenta, and with immediate ligature of the cord. When the cord was tied later, and the so-called "reserve" blood was expressed from the placenta, the percentage rose to one-ninth. In adults the relation of the blood to the body-weight is stated to be one-thirteenth. All authors agree that there is a temporary gain in the amount of blood when the cord is tied late.

Reaction.—The reaction of the blood at birth is always alkaline.

Color.—The color is found to be darker in the capillaries during the first few days than at any other time.

Specific Gravity.—At birth the specific gravity of the blood is about 1065, and this does not vary for the first few weeks. From this time up to the second year there is a constant diminution, decreasing in boys to as low as 1048, and in girls to 1050. It then gradually rises, till at the end of the first year it has reached the normal average of 1052 to 1055 (Hock and Schlesinger). The specific gravity does not seem to be influenced by the number of red or white corpuscles, food, rest, exercise, or other causes, but depends directly upon the amount of hæmoglobin. As a whole, the specific gravity is, apart from physiological variations, very constant in the same individual, and remains for weeks and months the same. Hock and Schlesinger place the greatest twenty-four-hour variation at 0.00025. The appearance of the child's skin is not an index to the specific gravity of the blood or to the amount of the hæmoglobin. Children often appear anæmic without any especial alteration in either of these conditions.

Hæmoglobin.—The hæmoglobin is found to be less firmly bound to the red corpuscle in the infant at term than it is in adults. It is, however, proportionately greater in amount at birth than in adult life. The hæmoglobin, like the specific gravity, which is closely dependent upon it, reaches its maximum at birth. Starting at 100 or 104, it falls rapidly to its minimum in the first three weeks of life (Hock, Schlesinger, Widowitz, Schmaltz, and Hammerschlag). The lowest percentage that has been found is about fifty-five per cent. From two weeks to six months it remains about the same and then rises slowly. The average figure for infants and children is sixty to eighty per cent. The percentage of hæmoglobin varies greatly not only between different individuals, but also in the same individual, which may explain the great discrepancy in the figures given by various authorities. Hæmoglobin is more abundant in boys than in girls.

RED CORPUSCLES (*erythrocytes*).—All authors agree that there is a large number of red corpuscles at birth, and also that an increase occurs in the first twenty-four hours. As regards the actual number in the cubic millimetre of blood there is much diversity of opinion. Hayem has found a larger number at the moment of birth than in the mother's blood, and gives as an average 5,350,000; Shiff says 5,800,000. The increase after birth is only seeming, according to Shiff, and is due to a loss of fluid and consequent concentration of the blood. From the second day the red cells begin normally to diminish, and fall eventually, according to Lepine, Jerrard, and Schlemmer, to 5,000,000. In infancy the average number is from 4,000,000 to 5,500,000; in later childhood from 4,000,000 to 4,500,000 (Hayem). There is much greater variation in size in the red corpuscles of the newly born than in those of older children or adults, and microcytes are more numerous; the oscillations in the number of red cells within twenty-four hours are also much more marked than in adults.

HÆMATOBLASTS.—Hæmatoblasts or nucleated red corpuscles are normally present in the fœtal blood and diminish in number towards the end of pregnancy. They also occur in the blood of premature infants, and are present in small numbers in the blood of normal infants for a few days after birth and then disappear. After six months they are rarely or never found normally. Pathologically their presence may be of considerable importance, and the younger the child the less intense need the anæmia be which will cause their appearance in the blood.

LEUCOCYTES.—The leucocytes, or white corpuscles, are more numerous at birth than in adults and in young children. In the first twenty-four hours the number varies from 18,000 to 30,000 (Schiff, Gundobin, Hayem). This is rapidly reduced at the end of ten days to from 14,000 to 10,000. The daily variations in the early days of life are more marked than in adults, and the leucocytosis of digestion is relatively higher. The average number of leucocytes in an infant's blood after the first two or three weeks and up to six months is from 12,000 to 14,000, and from six months to one year from 10,000 to 12,000. Denis places the number of white cells from the second to the sixth year at from 9000 to 10,000. Bouchut and Debrisay found the average number of counts in children from two to fifteen years of age to be 6700.

The following figures compiled by R. C. Cabot show the normal average number of blood-corpuscles at different ages in cases in which there was a loss of weight in the first forty-eight hours.

TABLE 73.

Normal Average Number of Blood-Corpuscles at Different Ages in Cases in which there was a Loss of Weight in the First Forty-eight Hours.

Age.	Erythrocytes.	Leucocytes.
At birth	5,900,000	21,000 (26,000 to 36,000 after first feeding.)
End of 1st day	7,000,000 to 8,800,000	24,000
" 2d "	generally increased.	30,000
" 4th "	6,000,000	20,000
" 7th "	5,000,000	15,000
10th day		10,000 to 14,000
12th to 18th day		12,000
1st year		10,000
6th year and upward		7,500

Five varieties of white corpuscles are found normally in human blood, and these are classified as follows by Ehrlich:

1. *Lymphocytes* (Plate XII., facing page 874).—These are about the size of a red corpuscle, and contain one large, round, deeply staining nucleus which entirely fills the cells. The protoplasm is not granular and stains faintly or not at all.

2. *Large Mononuclear* (Plate XII.).—These cells are much larger than the lymphocytes. They have one large oval nucleus with a broad margin of non-granular, almost colorless protoplasm about it.

3. *Transitional* (Plate XII.).—These cells are derived from the last form and are similar in size and color. The nucleus is indented on one side as the result of the beginning of nuclear division.

These three varieties are sometimes called basophiles. (Hewes.)

4. *Polynuclear Neutrophiles* (Plate XII.).—These are round cells, smaller than the large mononuclear, having a peculiar polymorphous deeply staining nucleus. The nucleus is long, irregular or twisted, and when stained often appears segmented. The protoplasm contains fine granules which are stained by neutral stains.

5. *Polynuclear Eosinophiles* (Plate XII.).—These cells are usually about the size of neutrophiles and have a deeply stained polymorphous nucleus. The protoplasm contains granules which are much coarser than those of the neutrophilic cells, and which stain readily with acid stains.

These cells are sometimes called oxyphiles. (Hewes.)

6. *Myelocytes* (Plate XII.).—These are round or ovoid cells with one, rarely two, large round or slightly bent nuclei which stain light blue. There are two varieties, neutrophilic and eosinophilic. The first has a protoplasm crowded with fine neutrophilic granules. The second contains coarse eosinophilic granules. Myelocytes rarely, if ever, appear in normal blood, but are much increased in some of the pathological states.

PERCENTAGES OF VARIOUS LEUCOCYTES IN NORMAL BLOOD.—Concerning the percentages of the different leucocytes in normal blood, it is sufficient to say that the blood of infants differs from that of adults in that the blood of the latter contains from 62 to 70 per cent. of neutrophiles, the remaining 40 to 25 per cent. being made up of mononuclear cells, of which about 28 per cent. are lymphocytes. The following figures illustrate this:

TABLE 74.

	Adults.	Infants.
Small mononuclear	20 to 30 per cent.	50 to 70 per cent.
Large mononuclear	4 to 8 "	6 to 14 "
Neutrophiles	62 to 70 "	28 to 80 "
Eosinophiles	½ to 4 "	½ to 10 "

Gundobin finds very little change from the above figures until the beginning of the third year, when the blood resembles more that of adults, the neutrophiles and mononuclear elements being present in about equal proportions. In children of eight or ten years he found very little difference from the blood of adults. His conclusions are that an infant's blood is (1) richer in white corpuscles; (2) richer in young form elements, the absolute and relative counts of the lymphocytes being three times as large as in the blood of adults, while the "overripe" elements, or neutrophiles, are half as many; (3) in infants the white corpuscles remain relatively longer in the "unripe," and in adults in the "overripe" stage.

Variations in temperature and physical exertion seem to have no effect upon the number of white corpuscles, but digestion leucocytosis is often quite pronounced. Most authors place the normal percentage of eosinophiles between 2 and 10 per cent. It is safe to say that they may be somewhat increased, even considerably, in infants' blood without having the same significance as in adults' blood.

THE BLOOD IN ABNORMAL CONDITIONS.

It is recognized that all the signs by which sickness is shown in the blood of adults are exaggerated in that of children. In my clinical work among infants I have noticed that their blood is much more sensitive to the action of adverse influences, such as poor air, improper hygienic surroundings, improper food, the inhalation of arsenic, and numerous other causes, and that anæmia is much more easily induced than in adults. This is especially noticeable in the impoverishment of the blood which follows gastro-enteric disease. Not only is an anæmic condition of the blood acquired by these influences, but also in congenital weaknesses which may be represented by infantile atrophy, or in some other inherited diseases, such as tuberculosis and syphilis, or in such acquired diseases as rhachitis, the development of the blood may be retarded. In this class of cases the blood of a child three or four years old may show no higher degree of development than that of a healthy infant in the second year. It is also found that when certain diseases occur in young children the blood is apt to revert to a more infantile condition, and that the normal leucocytosis of infancy may represent disease at a later period. Again, the erythrocytes under certain morbid conditions may revert to an earlier type, such as nucleated corpuscles. One of the most important points to be remembered is that a physiological leucocytosis depending upon digestion is commonly met with in infancy, and to a much greater degree than at a later period of life, so that what would be a normal leucocytosis at different times of the day, in reference to the food which has been taken, must always be borne in mind. It is therefore evident that when we are considering the blood of an infant or young child in any given disease we should take into consideration what the conditions of the blood usually show in the individual,—namely, as to whether a normal condition, according to the age, was present before the onset of the disease, or whether the especial individual had already an abnormal blood from backward development or from some inherited or congenital condition. Enlargement of the spleen in infants is not so significant as in adult life, for whenever anæmia is present there is apt to be an associated enlarged spleen.

A classification of diseases of the blood is as difficult to make and as unsatisfactory as that of infectious and non-infectious diseases. The one heading, however, under which all abnormal conditions of the blood may be brought is that of anæmia. It is probable that all the anæmic conditions of the blood will in the future be proved to be secondary, but when the causes of the pathological conditions found in the blood are difficult to find, or apparently are insufficient to cause so severe a disease as is present, we speak of primary anæmia, while we speak of secondary anæmias as those which have some obvious cause, such as rhachitis, malaria, syphilis, or hemorrhage.

SPECIFIC GRAVITY.—In various pathological conditions the specific gravity of the blood may be increased or diminished. Its changes usually run parallel with those in the amount of hæmoglobin, on which, in fact, it is largely dependent. It is diminished in all forms of primary and secondary anæmias, being most diminished in leukæmia and pernicious anæmia (1028). It is usually diminished in nephritis (1040) as a result of the small amount of albuminous substance in the serum. It is sometimes increased in cardiac affections, and especially in tubercular meningitis.

HÆMOGLOBIN.—In children the variation in the amount of hæmoglobin present from day to day is more marked than in adults, boys usually having more hæmoglobin than girls. It is diminished in secondary anæmia and in chlorosis, and lowest in leukæmia and pernicious anæmia (15 to 20 per cent.).

RED CORPUSCLES (*erythrocytes*).—A diminution in the number of red corpuscles is found in a large number of acute and chronic diseases, especially in those accompanied by a marked anæmia. Instances of these diseases are rhachitis, syphilis, tuberculosis, malaria, and chronic ileo-colitis. In leukæmia, and especially in pernicious anæmia, the number may fall to 500,000 per cubic centimetre. Certain drugs, such as arsenic, may in poisonous doses cause a marked diminution in the number of red cells. In recovery from acute anæmia the red cells regain their normal number much more rapidly than they do their hæmoglobin. The red corpuscle, which normally has a size of from six- to nine-thousandths of a millimetre, may undergo changes in size or shape, and cells much smaller (microcytes) or larger (macrocytes) than the normal red corpuscles may appear, also cells having oval, pointed, or irregular forms (poikilocytes). I have already spoken of the fact that nucleated red corpuscles are found normally in the blood of young infants. They also readily appear in quite large numbers in anæmia. There is, however, an essential difference in their significance in adults and in young children. In the former they only appear at a late period in severe anæmia, and therefore have a grave significance, while in children they readily appear in the blood in simple anæmias which go on to recovery.

WHITE CORPUSCLES (*leucocytes*).—The white corpuscles are affected in a temporary or permanent manner by many physical and pathological conditions in the body at large as well as in the blood-forming organs. The most common change from the normal is that of leucocytosis. Leucocytosis was originally described by Ehrlich as the presence in the blood of a greater number of white cells than normal for the individual, or a relatively increased number of any variety of white cells, with or without an increase in the total number of leucocytes. Cabot has restricted the term leucocytosis to an increase of leucocytes in which the polynuclear cells predominate, and uses the terms lymphocytosis and eosinophilia to describe a relative increase of the lymphocytes and eosinophiles. Physiologically, we find a leucocytosis after the ingestion of any proteid food. It is at its height about two hours after a meal, when the total number of leucocytes may be as great as from 13,000 to 30,000, according to the age of the child. Pathologically, a leucocytosis follows a considerable number of diseases, and seems in a general way to depend upon the amount of local reaction to which the disease gives rise. We find a pronounced leucocytosis in most fevers and in most septic processes. In these cases the increase is almost wholly composed of the polynuclear neutrophiles, which may make up from 90 to 98 per cent. of the entire leucocyte count. The leucocytosis does not depend on the degree of fever, does not always occur with it, and conversely inflammatory leucocytosis may appear before the fever. Pneumonia shows generally a leucocytosis. In pneumonia the large increase in the number of leucocytes seems to follow closely the course of the pathological process, and the "blood crisis" is found to anticipate the "temperature crisis" by some hours. Pericarditis and endocarditis, advanced pulmonary phthisis, pleuritis, erysipelas, acute rheumatism, purulent meningitis, pharyngitis, diphtheria, septicæmia, osteomyelitis, scarlet fever, variola, tonsillitis, bronchitis, peritonitis, acute nephritis, gastro-enteric inflammation, rhachitis (especially basophiles), some profound anæmias, whether primary or secondary, leukæmia, hemorrhage, malignant new growths, abscess of any kind, including appendicitis, and many skin diseases are among the affections that show leucocytosis. In leukæmia the leucocytes are increased more than in any other disease, they may reach 500,000 per cubic millimetre, and the proportion of the white to the red corpuscles may be 1 to 20 or even 1 to 6. Certain drugs cause leucocytosis, such as pilocarpine, antipyrin, thyroid extract, and tuberculin.

The diseases in which the leucocytes are approximately normal are malaria, tubercular meningitis, tubercular and serous peritonitis, influenza, measles, typhoid fever, pulmonary tuberculosis (unless there is a secondary infection by other bacteria), rubella, mumps, and most cases of epidemic influenza. Comparing these two lists it will be seen that there are instances in which the leucocyte count may be of great importance to the physician in making a differential diagnosis. By its aid we may in some cases differentiate scarlet fever from measles, a purulent from a tubercular meningitis, and a beginning pneumonia from a tubercular meningitis or typhoid fever. We may also differentiate typhoid fever from appendicitis, and from osteomyelitis, from ileo-colitis, and from septicæmia, also tubercular peritonitis from septic peritonitis. Lastly, we may by the leucocyte count alone be able to distinguish between sepsis and malaria in a patient whose only symptoms are malaise and returning chills.

LEUKÆMIA.

The disease called leukæmia sometimes occurs in infancy and childhood. Klebs, von Jaksch, and Sänger describe congenital cases. On the whole, it is a rare disease in infancy, and when it occurs it is probably always a mixed form. A pure myelogenous form of leukæmia is very rare. The etiology of the disease is obscure. Cases have been reported which followed congenital syphilis and rhachitis. It is thought by some to be an infectious disease, but the evidence is insufficient. Von Limbeck thinks that it is a disease of the lymphatic system. Others say that any anæmia or Hodgkin's disease may progress to leukæmia under certain circumstances, as may also anæmia infantum pseudo-leukæmica. A number of the cases are apparently primary. Males are more prone to the disease than females. The essential feature is a persistent increase in the number of the white corpuscles of the blood, associated with changes in the spleen, lymph-glands, or bone-marrow.

PATHOLOGY.—Leukæmia is a rare affection in infancy and childhood, and the recorded observations of these cases in early life are very incomplete. In the *splenic myelogenous* form the spleen is much enlarged. The condition of the organ is one of chronic hyperplasia, often with grayish-white lymphoid tumors scattered through it. The essential change in the bone-marrow is a great hyperplasia of the red marrow, which contains large numbers of nucleated red corpuscles in all stages of development, also myelocytes and other cells. In the *lymphatic* form of the disease there is a general enlargement of the lymphatic glands and other lymphoid tissues as a result of simple hyperplasia. This is usually associated with a moderate enlargement of the spleen. The liver, as a rule, is considerably enlarged in both forms of leukæmia.

Leukæmia is one of the few diseases which can be diagnosticated definitely from the blood examination alone. There is a more or less marked diminution in the number of the red corpuscles, the blood count ranging from 2,000,000 to 4,000,000, except in the early stages, with some irregularities in the size and shape of the corpuscles. The hæmoglobin is usually much diminished. The important change is in the leucocytes, which are enormously increased in number, the proportion being frequently 1 to 15 of the red, and sometimes even 1 to 3. In the *splenic myelogenous* form the chief point of interest is the great increase in the number of myelocytes, which average about 35 per cent. of the white corpuscles, and may be even 60 per cent. The eosinophiles are sometimes relatively increased. The percentage of polynuclear neutrophiles is usually diminished, and the lymphocytes show a great relative diminution. Many nucleated red cells are present. In the *lymphatic* form the character of the blood is materially different, and the increase in the number of leucocytes is not usually so great. The increase is due solely to the lymphocytes, all the other forms being greatly diminished relatively. Eosinophiles and nucleated red cells are rare. Myelocytes are not present or are rare. This description of the blood applies to children as well as to adults, but there are practically no complete data as to the character of the blood of leukæmias in infancy.

SYMPTOMS.—The onset is insidious, and usually the first striking signs are progressive enlargement of the abdomen, enlarged glands, shortness of breath, and general weakness or hemorrhages from the mucous membranes. The progress of the disease is usually much more rapid in children than in adults. The symptoms are similar in the two types of the disease. The lymphatic form of the disease is more common than the splenic myelogenous in infants and children. The child becomes gradually pale and weak, and suffers from dyspepsia and repeated diarrhœa. The respirations are accelerated, and dyspnœa is shown on slight exertion. Fainting attacks may occur. Hemorrhage from the nose is common, and may occur from the stomach, intestine, or kidneys, or into the skin. The pulse is usually rapid and soft. There are rarely any cardiac symptoms. Headache and vertigo are marked at times. Vision may be much impaired as the result of retinal hemorrhages or leukæmic plaques. Tenderness over the shafts of the long bones has been occasionally observed. The urine presents no constant changes. It occasionally contains albumin and casts. A moderate degree of fever is usually present in the late stages, also œdema of the feet and general anasarca.

The following table shows the average percentages of the different elements of the blood in the splenic myelogenous and lymphatic leukæmias:

TABLE 75.

Average Percentages of the Different Elements of the Blood in the Splenic Myelogenous and Lymphatic Leukæmias.

	Splenic Myelogenous Leukæmia.	Lymphatic Leukæmia.
Hæmoglobin	25 to 50 per cent.	25 to 40 per cent.
Red corpuscles	2,000,000 to 3,000,000	2,000,000 to 3,000,000
White corpuscles	100,000 to 400,000	50,000 to 100,000
Lymphocytes	10 per cent.	96 per cent.
Polynuclear leucocytes	50 "	3 "
Eosinophiles	5 "	rare.
Myelocytes	35 "	usually absent.
Nucleated reds	very numerous.	rare.

Diagnosis.—A distinction is chiefly to be made between leukæmia and Hodgkin's disease, or secondary anæmia with leucocytosis, which in infancy is often accompanied by an enlarged spleen. The difference between leukæmia and pseudo-leukæmia, if such can be said to exist, is explained on page 888.

The differential diagnosis of leukæmia is impossible by means of the clinical history, and can be made only by examination of the blood. The increase in the number of leucocytes is far greater, as a rule, in leukæmia than in other conditions, but the characteristic feature in the splenic myelogenous form is the presence of abnormal cells, namely, the myelocytes, and in the lymphatic form a great absolute and relative increase of the mononuclear cells. In Hodgkin's disease and in the secondary anæmias with leucocytosis there is a simple increase of the normal forms of leucocytes. In some cases of Hodgkin's disease also the blood may be normal. Chronic adenitis, which might be confounded with lymphatic leukæmia, shows usually normal blood, and is thus readily differentiated. In distinguishing between the two forms of leukæmia we rely much more on the quality of the blood than on the other clinical characteristics.

Prognosis.—The course is chronic and usually progressive, although recovery occasionally occurs. The great majority of cases prove fatal within a few months, although occasionally cases have been reported of three to five years' duration. Epstein and others have described an acute form with a rapid course of a few days or weeks. High fever, frequent hemorrhages, and marked œdema are unfavorable symptoms. The lymphatic cases run a shorter course and are more uniformly fatal than those of the splenic myelogenous type.

Treatment.—As in all cases of anæmia, fresh air and careful feeding are important indications. We have no specific for the disease, and the results of treatment by drugs of any kind are discouraging, although an occasional rare recovery has been reported. Arsenic given in large doses has proved the most efficient remedy. Iron is also of some value. Organotherapy has been used lately with some success. Bigger, de Feodosia, and Whait reported cases of rapid improvement after giving bone-marrow by the mouth. A glycerin extract of the spleen has been tried with little success. The results of splenectomy thus far indicate that it is not a desirable measure.

The following case of acute leukæmia occurred in the practice of Dr. Morse:

The child, a girl, was three years old and was healthy at birth. Her mother had died of pneumonia when she was two years old. Her father was well. There was no tubercular or syphilitic history. She was nursed for a year and then fed from the table. She was always subject to diarrhœa, but otherwise had been well. No history could be obtained except that six days previous to being seen blood-spots had appeared on the skin and the mouth had become sore. She had also vomited blood and had passed some blood from the bowels.

She looked seriously sick and was very pale, but was fairly developed and was not especially thin. Her gums were swollen, spongy, and bleeding, her tonsils were large, and her throat was reddened. There was a systolic murmur over the precordia. The lungs were normal. The abdomen was lax. The edge of the liver was felt about one inch below the costal border. The spleen was palpable below the border of the ribs to the extent of three inches in breadth and two inches in length. There was slight glandular enlargement. There were numerous petechiæ over the legs and abdomen. There were no evidences of rhachitis, no tenderness about the joints, nor swelling over the long bones. The temperature was normal. The blood count was as follows:

Hæmoglobin	25 per cent.
Red corpuscles	2,024,000
White corpuscles	87,400
Lymphocytes	83 per cent.
Large mononuclear cells	11 "
Polynuclear neutrophiles	5 "
Eosinophiles	1 "

There was a slight variation in the size of the red corpuscles, almost none in shape. There were no nucleated forms, and no myelocytes.

A fresh liquid diet, lemon-juice, and stimulants were ordered. The bleeding from the gums and bloody discharges continued, as did the purpuric efflorescence. The child was not seen again, but died a week later. There was no autopsy.

In the light of the above history and blood examination there can be no doubt as to the correctness of the diagnosis of acute lymphatic leukæmia. If the history can be accepted as correct, the onset was acute, there were subcutaneous hemorrhages and bleeding from the gums, and the duration of the disease was only two weeks. Owing to the circumstances of the child's surroundings, however, it is possible that these marked symptoms may have been preceded for a time by others less noticeable. The duration of the disease could not have been more than seven weeks. Although the glandular enlargement was not marked, that of the spleen was considerable. The great bulk of the white corpuscles was made up of lymphocytes. The large mononuclear forms were also somewhat increased in number.

The following case of splenic myelogenous leukæmia also came under the observation of Morse at the Infants' Hospital:

A boy, one year old, came to the out-patient department on April 26. His parents were Russians, and seemed well and strong. No history of syphilis or tuberculosis could be obtained. Four other children were alive and well, two had died of some acute disease in Russia. No very definite history of his illness could be obtained. He was weaned at three months and had been fed exclusively on condensed milk for six months. He had been failing gradually for some months. His abdomen had been large for two months, and he had been unable to lie on his left side for one month. He had never had epistaxis or hemorrhages from the stomach or bowels. He had never had any eruption on the body. He vomited occasionally. His bowels were somewhat constipated, and the dejections were light-colored.

The infant was markedly atrophic and anæmic. The head was large, the forehead prominent, and the anterior fontanelle widely open. There was a moderate rosary and an enlargement of the epiphyses, with slight bow-legs. There was marked enlargement of the glands of the neck, axillæ, and groins. The heart and lungs were normal. The abdomen was very much enlarged, but contained no fluid. The superficial veins were distended. The dulness of the liver began at the upper border of the fifth rib in the mammillary line, and the lower border could be felt round and smooth about two fingers' breadth below the costal border in the same line. The spleen was very much enlarged. It could be felt beneath the costal margin just outside the parasternal line, and just outside the umbilicus; it then extended beyond the middle line, and filled up almost the whole left iliac region. The notch could be plainly felt. The spleen and abdomen were not at all tender. There were hemorrhagic spots on the skin. The blood examination made at this time is given below.

It was impossible to keep the child under observation, but he was seen again on May 28. He had failed rapidly during the month. His skin was very pale with a decided yellow tinge, the face, body, and extremities being thickly covered with purpuric spots, varying in size from that of a pin's head to that of a lead-pencil. These spots had appeared two days before, at which time he developed a cough, and refused to eat. He had no other hemorrhages. The bowels were loose, the dejections were of normal color, and there was no vomiting. An examination of the chest showed a broncho-pneumonia involving the lower two-thirds of the left lung, and a well-marked bronchitis in the right lung. The heart was normal. The abdomen was rather more distended, was not tender, and there was no fluid. There was a slight increase in the size of both the liver and spleen. He was almost moribund.

The usual association of leukæmia with rhachitis was present in this case, the leukæmia having probably developed subsequent to the rhachitis. The latter was undoubtedly due to improper food and surroundings, and it seems more reasonable in the present state of our knowledge to consider the leukæmia as arising from the same cause rather than secondary to the rhachitis. The blood count made on April 26 showed 2,900,000 red corpuscles and 48,000 white corpuscles per cubic millimetre, giving a proportion of 1 to 60.

The differential count of one thousand white corpuscles on slips dried and stained with Ehrlich's triple stain resulted as follows:

Small lymphocytes	23.4	per cent.
Large lymphocytes and transitional forms	8.1	"
Myelocytes	21.4	"
Polynuclear neutrophiles	46.5	"
Eosinophiles	.6	"

A slip stained with eosin and methylene blue showed numerous mononuclear and polynuclear cells with fine granulations which took the eosinophile stain feebly. A slip stained with dahlia showed the presence of very few "mastzellen." Numerous partially destroyed cells, both mononuclear and polynuclear, were found.

There was a very marked poikilocytosis, a moderate number of microcytes, and numerous macrocytes, many of these being three times the diameter of a normal red corpuscle. Many of these forms lacked the normal concavity. Nucleated red cells were present in great abundance. No microblasts were seen. Many cells the size of normal corpuscles were met with in which the nucleus was smooth, feebly stained, and with little nuclear structure, the protoplasm being not quite homogeneous, "wrinkled," and stained a reddish violet. These may perhaps be regarded as undeveloped normoblasts. Normoblasts were very abundant, and always with one nucleus, which showed a well-developed nuclear structure, and which occasionally presented indentations as if division was beginning. The nucleus was rarely in the centre, often on the side, sometimes half-way out of the cell and occasionally free. The protoplasm was ordinarily narrow, but sometimes wide. A few cells were met with from which the nucleus had probably escaped. Megaloblasts were quite numerous and presented the same variations as the normoblasts. In addition to these a number of large oval cells were seen which contained two nuclei, the long diameter of the cells being about four times that of the normal red corpuscles. In several of these well-marked karyokinetic figures were present; these variations in the erythrocytes, of course, merely showed a severe grade of secondary anæmia.

PSEUDO-LEUKÆMIC ANÆMIA OF INFANCY.

Von Jaksch in 1889 and 1890 was the first to describe this disease and give it its title. Since then it has been the subject of much investigation and contention, and there appears to be a great difference of opinion among those who use the term as to what constitutes the disease. Von Jaksch based his diagnosis on the following points. (1) It is a disease of infancy whose symptoms and course correspond to the picture of leukæmia. (2) It is characterized by a marked reduction in the number of red corpuscles and in the hæmoglobin, and a marked leucocytosis, a great enlargement of the spleen with only a moderate or slight enlargement of the liver, and with at times enlarged lymphatic glands. It is clinically differentiated from leukæmia by the disproportion existing between the size of the liver and the spleen. The course of the disease is less rapid and the prognosis better than in leukæmia. Luzet, Alt, and Weiss give the following additional description of the blood. There is marked irregularity in the size and shape of the red cells. Many nucleated red cells, normoblasts and megaloblasts, many with dividing nuclei (karyokinesis), and many polychromatophilic red cells are present. The karyokinesis is regarded by some as characteristic of the disease. The leucocytosis is a polymorphous one. The eosinophilic cells vary in number and are at times much increased.

Etiology.—This disease is found in infants usually between the ages of six and twenty months. It is more frequent in girls than in boys. Von Jaksch considers it a primary condition. Fischl and Epstein believe it may be secondary to many conditions, such as septicæmia, tuberculosis, intestinal infections, and syphilis. In the twenty cases reported by Monti and Berggrün, sixteen had a history of syphilis, and one of rhachitis. Monti says it may develop from cases of severe anæmia with leucocytosis and believes it may terminate in leukæmia.

Pathology.—The spleen is always enlarged; it may attain to from four to sixteen times the normal weight, and extend to the crest of the ileum.

It is firm, with rounded edges, and shows microscopically a simple hypertrophy of the spleen pulp. The liver is enlarged in about half the cases, and much less in proportion than the spleen. Histologically it has the character of the fœtal liver. The lymph-glands have been moderately enlarged in about half the reported cases. The bone-marrow was found to be congested in some cases.

Symptoms.—The chief features of the blood in pseudo-leukæmia have been already noted. The reduction of the hæmoglobin often reaches as low as 30 per cent. The specific gravity ranges between 1040 and 1035. The proportion of white to red cells is usually 1 to 100 or 1 to 75, in severe cases reaching 1 to 20 or 1 to 12. The red cells usually number between 2,000,000 and 3,500,000, sometimes 1,000,000 or less. The leucocytosis ranges from 30,000 to 100,000.

The onset is always insidious, sometimes with gastro-enteric disturbance and the usual signs of anæmia; marked pallor and weakness develop slowly. The child becomes apathetic, but does not appear to suffer, and often remains plump and well nourished. Sometimes a slight œdema of the dependent portions of the body appears. The abdomen becomes prominent, and sometimes uniformly distended, again only in the region of the spleen, which becomes very large and easily palpable. Digestion is impaired. The urine is usually normal, but occasionally a slight albuminuria appears. There may be occasional elevations of temperature, even in the absence of any complication. The course of the disease is a chronic one, lasting several months or a year, not longer. It is not necessarily fatal; recoveries have been reported by von Jaksch, Monti, Comby, and others. The infants are so feeble, however, that they are very apt to be carried off by intercurrent diseases. Cases have been reported by various authors which apparently developed from an ordinary severe simple anæmia, and others have been noted in which the blood gradually developed all the characteristics of leukæmia. In fact, certain observers have regarded the pseudo-leukæmic anæmia of infants as an early stage of leukæmia.

Diagnosis.—This disease is to be especially distinguished from simple anæmia with leucocytosis on the one hand, and from leukæmia on the other. In the first the leucocytosis is smaller and the splenic enlargement much less. In leukæmia the leucocytosis has a different character, and there is less reduction in the red cells and in the hæmoglobin; there is also a proportionately greater hepatic enlargement and a relatively unfavorable course.

With reference to the status of this disease, it is still an open question whether the pseudo-leukæmic anæmia of infants can be considered as a distinct disease. The descriptions of the disease given by various authors are far from being identical; in fact, they vary so much that it is hard to see why the same title is given to them. Some authors consider it always primary, and would rule out all cases developing with rhachitis, syphilis, or tuberculosis, while others would include them. Among the cases reported under this name have been unquestionably included some of leukæmia, pernicious anæmia, and secondary anæmia with leucocytosis. The condition of the red cells is not characteristic. It may occur in any severe anæmia. The leucocytosis is not characteristic of the disease, it is simply a question of degree. Cases of ordinary secondary anæmia which have been closely observed appeared to develop into pseudo-leukæmic anæmia, and von Jaksch claims to have seen pseudo-leukæmic anæmia develop into a leukæmia. In short, it is held by leading authorities, such as Raudnitz, von Limbeck, Ebstein, and Fischl, that there is nothing in the etiology, clinical history, or blood to justify its classification as a separate disease, and that the majority of the cases reported under this title are simply severe types of secondary anæmia. We must look to the future to decide these questions.

PROGNOSIS.—The mortality has not been over 20 per cent. The prognosis should always be guarded, but not hopeless, and if the leucocytosis remains moderate and no complications occur, an arrest of the morbid process may be expected.

TREATMENT.—The combination of arsenic with iron is most valuable in this disease. The arsenic is best given in the form of Fowler's solution in drop doses repeated several times a day after feeding. The general treatment is the same as in secondary anæmia.

The following case of pseudo-leukæmia was treated in my wards at the Children's Hospital:

A boy, three years of age, had never had any disease, with the exception of a questionable malaria, from which he had entirely recovered two years previously. Since his second year he had been pale. He entered the wards on October 4. He was then of a waxen color, and the mucous membrane of the lips and nails was nearly white and had a livid tinge. The skin was almost translucent. There was not much emaciation. The spleen was considerably enlarged and could easily be felt about two inches below the border of the ribs. The liver was slightly enlarged and palpable. The glands in the neck, axillæ, and groins were enlarged to the size of peas. On percussion the heart showed no enlargement. A loud systolic murmur was heard over all the cardiac orifices. The action of the heart was very rapid but regular. Its impulse was in the fifth interspace inside of the mammillary line. Auscultation and percussion of the lungs showed that they were normal, with the exception of some sibilant râles. The respirations were 30 to 44 in a minute. The temperature at entrance was 38.3° C. (101° F.), and afterwards varied from 39.5° C. (103.8° F.) to 38.3° C. (101° F.). The pulse varied from 125 to 150. Diarrhœa was present when the child entered the hospital, and at first there were from four to six very offensive movements daily. For three or four days preceding death the movements were more frequent, but not so offensive, and contained mucus. Vomiting occurred at times. The infant was treated with modified milk, bismuth, and stimulants. An examination of the blood, October 13, resulted as follows:

Red corpuscles	1,295,000
Hæmoglobin	15 per cent.
White corpuscles	64,500

There were numerous poikilocytes, microcytes, and megalocytes. A number of the corpuscles were pale and many of them contained very little hæmoglobin. There were numerous nucleated red corpuscles, chiefly normoblasts, and in many of them the nuclei were undergoing subdivision. The eosinophiles were absolutely and relatively increased. No myelocytes were present, and the leucocytes were largely of the polynuclear form. The child died October 20 and a partial autopsy was obtained. A microscopic examination showed no evidence of leukæmia in the liver, spleen, kidneys, or lymph-glands. There was no evidence of syphilis or rhachitis, nor of any inflammation which could have caused the leucocytosis.

PRIMARY ANÆMIAS.

Pernicious Anæmia.—This is the most severe form of primary anæmias, and is characterized by quite constant changes in the blood, usually developing without evident cause, and a progressive course, leading to a fatal termination in the majority of cases.

ETIOLOGY.—This disease is much less frequent in children than in adults, the proportion of cases being about one to seventeen. It is less common in infancy than in later childhood. Of twenty-five reported cases, four were between the ages of three months and two years, four between three and five years, eight between six and ten years, and nine between ten and fifteen years. In the majority of cases no definite cause has been discovered. In some, the following etiological factors have been noted: syphilis, severe rhachitis, especially with splenic enlargement, and intestinal parasites, such as tænia, ankylostoma, and bothryocephalus. The best theory for the explanation of the condition seems to be that of a toxic hæmolysis. There are evidences of increased destruction of blood-cells in the urine, blood, and tissues, and also of a compensating hypertrophy in the blood-forming organs. There is considerable evidence that the alimentary tract is the source of the unknown toxins causing hæmolysis.

PATHOLOGY.—The body is not often emaciated. The skin usually shows a lemon-yellow tint. There is marked anæmia of the organs at times, with many capillary hemorrhages into the organs and skin. The heart is large and flabby, and shows intense fatty degeneration. The liver is fatty and sometimes enlarged. The kidneys and blood-vessels show fatty changes. One of the most characteristic features is a deposit of iron in the liver tissue and often in the intestinal mucosa. The spleen is hard and somewhat increased in size as a result of overgrowth of the fibrous tissue of the pulp. Some of the lymph-nodes are often enlarged. The bone-marrow is "splenified,"—that is, returned to its embryonic state.

SYMPTOMS.—*Blood.*—The red corpuscles are much diminished, usually to one million, sometimes even to five hundred thousand. The hæmoglobin is diminished per bulk of the blood, often to twenty-five or thirty per cent., rarely to twelve or fifteen per cent., but owing to the greater reduction of red cells it is often relatively increased per corpuscle (the color index is high and sometimes greater than normal). This is exactly the opposite condition to that which occurs in chlorosis. The red cells show great variation in shape and size, many oval or rod-shaped or irregular corpuscles are present, which take stains in an uneven way. A majority of the red cells are larger than normal (megalocytes). Many nucleated red cells are present, both of normal size (normoblasts) and of larger size (megaloblasts). The megaloblasts are the more numerous of the two. Many of the nuclei are found in a process of division (karyokinesis). The leucocytes are normal in number, or somewhat diminished,—eight thousand to four thousand. In the more severe cases there is frequently a relative increase of the lymphocytes with a corresponding diminution of the polynuclear cells. A few myelocytes are not infrequently found.

The general symptoms are those of severe anæmia, increasing pallor and prostration, usually without emaciation. The onset is insidious, and the clinical course does not differ from that of adults. There is frequently more or less gastro-enteric disturbance, vomiting, nausea, or dyspnœa. Hemorrhages occur into the skin or from the nasal and other mucous membranes. Œdema often appears in the dependent portions of the skin, less frequently in the cavities of the body. Hæmic murmurs are usually heard in the cardiac region. The pulse is full and soft. It is to be noted that hæmic murmurs are less frequent in the anæmia of infants than in that of adults. The urine is scanty and of low specific gravity. The temperature is variable; at times it may be normal, and at others an irregular pyrexia may run for a considerable period. The course is a chronic one, but varies in length. The disease may be completely developed in some cases in two or three months, and in others only after ten or twelve months. In most instances it progresses steadily to a fatal termination.

DIAGNOSIS.—The diagnosis from other forms of anæmia is made chiefly by the blood examination. The characteristic features are:

(1) Great diminution in the number of red cells.

(2) Relative increase of hæmoglobin per corpuscle,—high-color index.

(3) Irregularity of distribution of hæmoglobin in the red cells.

(4) Marked irregularity in form and size, and a general increase in size of the red cells.

(5) An excess of megaloblasts over normoblasts.

(6) A normal or diminished leucocyte count.

PROGNOSIS.—The prognosis is almost absolutely fatal, except in those cases associated with intestinal parasites or rhachitis, although lately, when arsenic has been given, the proportion of recoveries has increased. The blood examination is a valuable index to the prognosis. The presence of many normoblasts is hopeful, pointing to a regeneration of the bone-marrow. The presence of a large excess of megaloblasts indicates a more malignant case.

TREATMENT.—Arsenic is more useful than iron in this form of anæmia.

It is usually well borne, and should be given in small doses, gradually increased to the limit of tolerance. A child of ten years can usually take 2 c.c. (30 drops) of Fowler's solution in the twenty-four hours. Arsenic may also be given in pill form. Active exercise should be prohibited and replaced by massage, which is of great value and never to be neglected. Rest in bed, either absolute or for the greater part of the time, is essential. Small, frequent, and regular meals should be given, of light, nutritious food. Bone-marrow in the form of a glycerin extract has been recommended, but has thus far proved of doubtful value. The free administration of oxygen day and night for several weeks is worthy of trial, if expense is not to be considered. I have had one case in which the red cells had fallen to 1,088,000 and the hæmoglobin to 20 per cent. Rapid improvement in the blood followed this treatment when carried out for over four weeks, and the blood eventually became normal. The case is still under observation.

The following case, probably one of pernicious anæmia, though the result of the blood examination is not conclusive, was seen in consultation with Dr. C. P. Putnam.

The infant was healthy at birth, and up to the time of its present sickness had never had any disease. For several months it had grown progressively pale, its appetite had decidedly lessened, it had not lost materially in weight but had grown weak, and its mental hebetude had been so noticeable that a suspicion had arisen that it was lacking in cerebral development. On inspection the infant seemed moderately fat, but the muscles were soft and the skin was of an extremely pale and waxen tinge. It was evidently very weak. On physical examination nothing abnormal was detected about the head, thorax, or abdomen. All the organs seemed to be of natural size. The blood examination showed the following:

Red corpuscles	1,571,000
Hæmoglobin	22 per cent.
White corpuscles	19,100
Small mononuclear	42 per cent.
Large mononuclear	18 "
Polynuclear	40 "

The child died a few days later without showing any other symptoms.

Chlorosis.—Chlorosis is a primary anæmia of unknown cause, characterized by a marked diminution in the hæmoglobin in the blood without a corresponding loss in the number of red cells. It is not a disease of infancy or of young children, but appears in girls, especially blondes, between the ages of twelve and seventeen years. The essential cause is unknown. It is chiefly seen in overworked girls living amid poor hygienic surroundings. Psychical disturbance, such as grief, worry, and fright, are important factors. Clark has stated that the condition is a copræmia due to toxic absorption (ptomaines and leucomaines) from the intestine.

Pathology.—Chlorosis is rarely fatal. In some cases hypoplasia of the heart, large vessels, ovaries, and uterus have been found, also considerable fatty degeneration of the heart and of the intima of the vessels. Gastric ulcer has been occasionally associated with it.

Symptoms.—The essential change in the blood is a marked loss, in the individual red corpuscle, of the hæmoglobin, which is often only 40 per cent., and in severe cases below 30 per cent. The specific gravity of the blood may be reduced to 1025, although usually not below 1035. The red cells in a few cases are normal; in the majority they are reduced to 4,000,000 or may be even lower. The red corpuscles may be very irregular in size and shape in severe cases, and may have an average size which is less than normal. Nucleated red cells are often seen, usually normoblasts, rarely megaloblasts. Leucocytosis is not present. The onset of chlorosis is gradual, the course chronic, and relapses are common. The symptoms are those of a simple anæmia. The patient complains of shortness of breath on exertion, and attacks of palpitation and syncope are common. The appetite is poor or perverted, and there is a craving for unusual and indigestible articles of diet. Gastric hyperacidity is often present, and constipation almost always. Enteroptosis is not uncommon. The patient frequently complains of headache or neuralgic pains. Hysterical manifestations may occur. The menstrual function is often disturbed; the flow is scanty, irregular, and sometimes painful. The patient is usually plump and soft, and the skin has a peculiar greenish-yellow tint which gives the disease its popular name of "green sickness." Systolic murmurs are frequently heard over the heart in the pulmonary or mitral region, and also a venous hum, the "bruit de diable," in the veins of the neck. Palpitation is occasionally seen in the jugular veins. There is a tendency to venous thrombosis, which most often occurs in the femoral veins. The pulse is full and soft. Occasionally there is some puffiness of the face, and often œdema of the ankles. The spleen may be slightly enlarged. The urine rarely contains a slight trace of albumin. Gastric ulcer sometimes occurs as a complication.

Diagnosis.—The age and sex of the patient, the well-nourished condition, the pearly sclerotics, the color of the skin, and the character of the blood form a striking clinical picture which easily distinguishes chlorosis from other forms of anæmia.

Prognosis.—The prognosis is almost always good. The disease often lasts a year and relapses are common. Chlorosis is rarely fatal, except when complicated by tuberculosis or gastric ulcer.

Treatment.—There are few therapeutic measures in medicine more satisfactory than the use of iron in chlorosis. In infants the iron can be given in the form of the saccharated carbonate, 0.12, 0.18, or 0.24 gramme (2, 3, or 4 grains), or of the tartrate of iron and potash. Lactate of iron can also be given to older individuals in doses of 0.06 to 0.12 gramme (1 to 2 grains), gradually increased to 0.24 to 0.36 gramme (4 to

6 grains), three times a day. The administration of iron is usually followed by a rapid increase in the number of red cells to the normal or even above it. The hæmoglobin, however, increases more slowly. When iron is not tolerated, arsenic can be given in the form of Fowler's solution. When the iron causes constipation, it should be accompanied or followed with a saline or with some preparation of rhubarb. A change of scene is often beneficial, and later an out-of-door life and moderate exercise.

SECONDARY ANÆMIAS.

Under the title of secondary anæmia we include anæmia resulting from many kinds of pathological processes in the body which occur outside of the blood-forming organs.

Etiology.—Almost every disease of any organ in the body may produce a secondary anæmia, and it is of high or low grade according to the severity of the disease. There are two important factors in the process,—namely, an insufficient production of blood on account of poor food or poor assimilation, and the increased destruction of blood as it occurs in many wasting diseases. The anæmia of infancy may be either congenital or acquired. The congenital forms are inherited from delicate or anæmic parents, or from a mother who has suffered with syphilis, tuberculosis, or malaria during her pregnancy. Of the acquired anæmias, a comparatively small number of cases occur after hemorrhage, as in epistaxis, hæmophilia, purpura, or scorbutus. The more frequent causes of anæmia in infants and young children are disturbances of digestion and assimilation, such as are represented by inanition and chronic indigestion resulting from improper quantity and quality of the food. Anæmia may also result from chronic diarrhœa, ileo-colitis, constitutional conditions resulting from unhygienic surroundings, bad air, close confinement, general debility, rhachitis, and also from malignant disease. Another cause is found in the drain on the albumin of the blood occurring in chronic suppuration, nephritis, and effusions, and still another in the toxæmia produced by mineral poisons, such as arsenic or lead, or by infectious diseases, such as septicæmia, diphtheria, scarlet fever, syphilis, malaria, and tuberculosis.

Symptoms.—Luzet divides the anæmias of infancy into two classes,—those with splenic enlargement and those without,—and states that the first form is more severe, and that the blood in these cases contains a larger number of nucleated red cells. This classification is not a very satisfactory one, for we find very severe anæmias without splenic enlargement, and also marked splenic hypertrophy in many cases of mild anæmia. Monti and Berggrün propose another classification, which is more satisfactory. They divide all secondary anæmias into four classes:

(1) *Mild anæmias.*
(2) *Mild anæmias with leucocytosis.*
(3) *Severe anæmias.*
(4) *Severe anæmias with leucocytosis.*

In the mild anæmias there is a moderate diminution in the specific gravity of the blood, in the hæmoglobin, and in the red cells, and the latter show practically no changes in size and in shape. In the severe anæmias there is a marked diminution in the specific gravity, hæmoglobin, and red cells, and the latter show considerable changes in size and form (poikilocytes, normoblasts, megaloblasts). The difference between these two types is one of degree. Either form may be associated with leucocytosis, and the forms with leucocytosis are much more common than those without, and are almost invariably the more severe. In all but the mild anæmias without leucocytosis the spleen may be enlarged. The number of red cells varies from normal, in the mild cases, to two million in the severe, the specific gravity from 1056 to 1035, the hæmoglobin from eighty to thirty per cent., the white count from normal to sixty thousand. In secondary anæmia the reduction in the total amount of hæmoglobin is almost always proportionately greater than in the corpuscles (chlor-anæmia). In general, the more severe the anæmia the lower is the specific gravity of the blood, the greater is the variation in size and shape of the red cells (poikilocytosis), and the more numerous the nucleated red cells. These last are much more abundant in children than in adults with a corresponding grade of anæmia. In these cases we have the familiar clinical picture of pallor, soft muscles, digestive disturbance, fretfulness, headaches, insomnia, fatigue, and breathlessness on exertion. Occasionally epistaxis is associated with the signs and symptoms of the primary disease. Hæmic murmurs are usually heard over the heart and the vessels of the neck. Œdema is more common in older children than in adults.

Diagnosis.—In the diagnosis of these anæmias the recognition of the cause is very important. The diagnosis from the so-called primary anæmias is sometimes quite simple, by means of the blood examination, especially in older children; in young children it is often very difficult, as a result of certain factors which have already been mentioned,—namely, the frequency of splenic hypertrophy in all forms of anæmia, the ease with which a leucocytosis is developed, the frequent occurrence of abundant myelocytes and nucleated red cells, and the fatality of all severe anæmias in children; in short, the exaggeration of all blood changes in infants and young children.

Prognosis.—The prognosis depends on the age, cause, and condition of the blood. The course of the anæmia is usually several months. Traumatic cases, which are rare in childhood, are the most benign, since in these cases the blood is readily restored to its normal condition. The group of cases which depend on toxæmia or a drain of the albumin of the blood, such as in suppuration, fever, or from other causes, are difficult to treat, unless the cause can be removed. The severe cases may develop into a pernicious anæmia or a leukæmia, but death occurs more commonly as a result of some complicating disease rather than of the anæmia itself. The prognosis is naturally less favorable in cases in which the hæmoglobin is reduced to 30 per cent. or less, and the red cells to half their normal number, with marked poikilocytosis. The cases with leucocytosis are graver than those without.

TREATMENT.—It is of the first importance to treat the cause, as, for instance, cases which are secondary to malaria, syphilis, or rhachitis; or to remove the cause, if possible, in cases of inhalation or ingestion of poisons, such as arsenic, impure air, or improper food. The general treatment in infants and young children is usually far more important than the use of drugs. The treatment of infants is, above all, prophylactic, with the object of protecting them from such unfavorable influences as may interfere with their health and proper development. The question of feeding or of adapting the food to the child's digestion is of great importance, and fresh air, in the country if possible, is indicated. The best forms of iron to be used in the secondary anæmias of infants and young children are the saccharated carbonate and the tartrate of iron and potash. At times arsenic in the form of Fowler's solution can be used.

The following case of secondary anæmia, occurred in a child suffering from rhachitis with splenic enlargement, and is represented in Fig. 86, page 337.

The child was three years old and fairly well nourished. It had, however, enlarged epiphyses, a rhachitic rosary, a square, rhachitic head, and marked bowing of the legs. On physical examination there was no indication of enlargement of the liver or glands. The spleen was very much enlarged, its outline showing the notch as indicated in black. The blood examination gave the following result:

Red corpuscles	2,686,250
Hæmoglobin	35 per cent.
White corpuscles	13,000

There were poikilocytosis and a marked pallor of the corpuscles. (Wentworth.)

The following is a case of secondary anæmia occurring in connection with congenital syphilis with enlarged spleen, and is represented in Fig. 121, page 536.

The infant was three months old, and was nursed by its mother. It was healthy at birth, and remained so until three months old, when it showed marked syphilitic symptoms, which later became very characteristic. It was fairly well nourished. The skin was of the waxen pallor characteristic of the higher grades of grave anæmia. There was a moderate enlargement of the liver, which, on palpation, was found to be hard and somewhat tender. The inguinal lymph-nodes were slightly enlarged. The post-aural lymph-nodes were enlarged. The spleen was much enlarged, and extended from the fifth rib to the left inguinal region. It had a peculiar tongue-shaped outline, and was hard but not tender. There were no other glandular enlargements. An examination of the blood gave the following result:

	Nov. 17.	Nov. 20.
Red corpuscles	3,387,000	3,300,000
Hæmoglobin	47 per cent.	45 per cent
White corpuscles	20,000	20,000

There was considerable variation in the size of the red blood-corpuscles, which were pale and showed a moderate degree of poikilocytosis; there were also some microcytes and megalocytes. The mononuclear elements predominated. The eosinophiles were not numerous. (Wentworth.)

THE LYMPH-NODES.

Pathological conditions of the lymph-nodes may be primary or secondary. The *primary forms* are represented by simple inflammations of the lymph-nodes (*simple acute adenitis*), caused by the colon bacillus and other micro-organisms from the mouth and gastro-enteric tract. Among these organisms the tubercle bacillus holds an especially prominent place.

The *secondary forms* are due to tuberculosis, syphilis, and anæmia, and have been considered under these diseases.

SIMPLE ACUTE ADENITIS.

Etiology.—The place of invasion of the infection which produces simple acute adenitis is either the skin or the mucous membranes, the bacteria or their toxines reaching the nodes by means of the lymphatics.

The *cervical nodes* may be infected from a number of sources, such as irritation or lesions of the scalp, the ears, eyes, nose, throat, gums, or teeth.

The *bronchial nodes* may be infected from inflammation of the trachea, bronchi, and lung tissue, such as in bronchitis and pneumonia.

The *mesenteric nodes* may be infected from such conditions of the intestine as cholera infantum and ileo-colitis.

Axillary adenitis may result from lesions of the arm, such as are produced by vaccination; and in a like manner *inguinal adenitis* my follow lesions of the foot and leg or inflammatory conditions of the genitals.

Acute adenitis is also a frequent complication of the acute infectious diseases, especially scarlet fever, measles, and diphtheria.

Infection and enlargement of the superficial nodes demand special consideration, as they are of frequent occurrence and are easily detected. Inflammation of the deeper nodes had best be described in connection with the organs with which they are especially connected, as their clinical recognition is so often impossible. Thus, when there is an acute inflammation of the bronchial or mesenteric nodes, general symptoms of cough and diarrhœa are usually referred to the lung or intestine, unless, as may occur in extreme cases, distinct symptoms of pressure in the thorax or palpable tumors in the abdomen are present.

Pathology.—The affected nodes show a condition of acute congestion and hyperplasia, but do not become cheesy, except when the infection is tubercular. The process may end in suppuration or resolution, according to the severity of the infection. Suppuration is especially frequent in infants and young children, and the pus usually begins in the second or third week from the onset of the morbid process.

Periglandular cellulitis is common when the inflammation is acute and severe.

easily from Hodgkin's disease, but in the latter the enlargement of the nodes is greater and more generally distributed, the secondary anæmia is more pronounced, and the disease is of rare occurrence as compared with chronic adenitis.

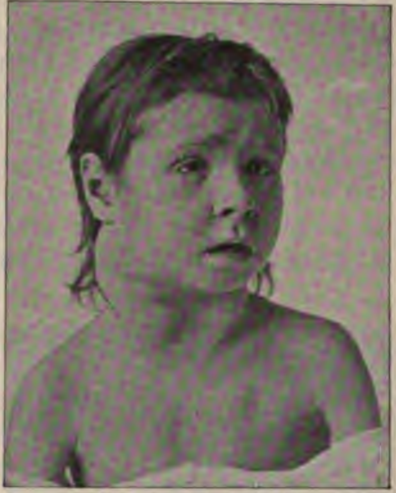
From Syphilitic Adenitis.—The absence of other signs of late syphilis, such as are described on page 536, and the negative results of anti-syphilitic treatment make the exclusion of syphilitic adenitis comparatively easy.

From New Growths.—New growths are usually to be distinguished by their greater size and by the absence of an exciting cause, more positively by the microscopic examination of a portion of the node excised for the purpose of diagnosis.

TREATMENT.—In the treatment of simple chronic adenitis, the primary exciting cause should be removed if possible. Local applications should be avoided, and general tonic treatment instituted by means of cod-liver oil, Fowler's solution, the iodide of iron, good food, and out-of-door life. If these measures fail, the treatment becomes surgical.

Fig. 185 represents a case of chronic cervical adenitis in which the cervical lymph-nodes were enlarged to such an extent that they had become a deformity. Nothing else abnormal nor any symptom of tuberculosis was discovered about the child.

FIG. 185.



Chronic cervical adenitis.

DISEASES OF THE THYROID GLAND.

The function of the thyroid gland is not definitely known, but it is generally believed to secrete certain nutritive or antitoxic substances which are necessary for normal metabolism. Disturbance in the function of the gland, resulting either in hypersecretion or diminished secretion, gives rise to different types of symptoms. Such disturbances may be produced by simple hyperæmia, hypertrophy, atrophy, new growths, or as a result of excision of the gland.

GOITRE (Bronchocele).

Enlargement of the thyroid gland is commonly called goitre. True goitre consists in the enlargement of the old and in the formation of new alveoli, in the cells of which a greater or less amount of colloid degeneration takes place. The colloid abnormalities of goitre are rarely present in children (Rex), in whom the thyroid enlargement seems to be little becoming more prominent. The catamenia had not appeared. The girl was well and strong, but was more fretful and capricious than seemed consistent with her usual temperament. The tumor was elastic, did not fluctuate, and was neither red nor tender.

MYXŒDEMA.

Myxœdema is a constitutional affection, generally associated with atrophy of the thyroid gland, and characterized clinically by a thickened and dry condition of the skin and subcutaneous tissues, and later by mental failure.

The symptoms are supposed to be caused by a diminished or total lack of secretion of the thyroid gland due to its atrophic condition (*athyrea*). The disease may be *congenital* or *acquired*. Acquired myxœdema is seen most commonly in adults, and will not be especially described. *Operative myxœdema*, or *cachexia strumipriva*, is a condition closely resembling acquired myxœdema of adults, and follows the removal of the thyroid gland by operation. The form of myxœdema which is especially related to infants and children is that which is known as *cretinism*, or congenital myxœdema.

Cretinism (*congenital myxœdema*).—Two classes of cases are recognized: one, known as *endemic cretinism*, represented by the cretins of Switzerland, Italy, France, of the great lakes (Michigan) of America, and of other places; and a second class, known as *sporadic cretinism*, represented by individual cases which may be met with anywhere.

PATHOLOGY.—There may be a congenital absence of the thyroid gland, or atrophy may occur, as after one of the specific infectious diseases.

The head in sporadic cretinism is usually brachycephalic,—that is, it is contracted in its antero-posterior diameter. Virchow was the first to observe in these cases a premature ossification of the spheno-basilar bone. The sphenoid and basi-occipital bones should remain separate until about the fifteenth year, and their early ossification explains, according to Virchow, the changes which take place in the form of the cretin skull and face.

Occasionally the thyroid gland in a cretin is enlarged, as in a simple goitre, but its function is absent, as shown by the symptoms characteristic of athyrea.

There are certain characteristics of the bones in cretins, consisting of an enormous overgrowth of cartilage, an arrest of growth at the distal ends of the bones, and a premature ossification of the shaft. Fig. 82, II., page 329, represents a section of the tibia of a sporadic cretin child. It is distinguished anatomically by an almost entire absence of the zone of proliferation (Whitney).

SYMPTOMS.—In sporadic cretinism the symptoms of the disease usually develop in the first year of life, sometimes not until the second or third year or even later. The body is dwarfed. The anterior fontanelle remains open until the eighth or tenth year. The face is full, stolid, and expressionless. The nose is broad and flat; the eyes are small, wide apart and half closed by the swollen lids. The mouth is open, the lips are thick, and the tongue protrudes. The cheeks are swollen and bloated. The hair is coarse and straight, and usually light-colored. The teeth are rough, irregular, and liable to early decay. The voice is rough and harsh. Spinal curvature is apt to be present, and is much more apt to be in the nature of a lordosis than of a kyphosis. The extremities are short and stunted, and the hands and feet are pudgy. Normally at birth the legs are about 43 per cent. of the total height, and at four or five years about 50 per cent. In cretins this percentage is lessened to between 35 and 40 per cent. of the total length. The skin is everywhere dry, thick, and bloated, and does not pit on pressure. Perspiration is scanty. The muscles are weak and flabby, and the motions slow and clumsy. The neck is short and thick, and the abdomen large and pendulous. Fatty tumors are not infrequently to be found in the supra-clavicular regions. The genitals are small and undeveloped. Habitual constipation is common. The temperature is frequently subnormal. The temperament of cretins is placid, and their expression stupid or even idiotic. Their mental and physical development is very backward; they learn to talk late or not at all.

DIAGNOSIS.—The appearance of a cretin is so characteristic that a diagnosis is rarely difficult. Fœtal or early and severe rhachitis may give rise to marked deformities suggestive of cretinism, but do not show the other physical signs and the mental symptoms.

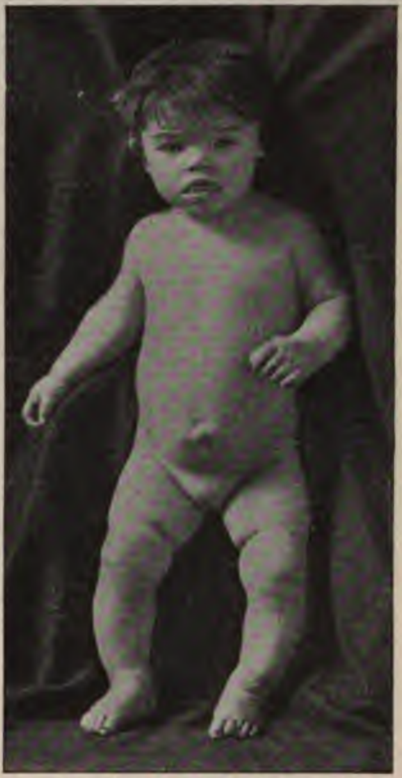
TREATMENT.—The recognized specific treatment of all forms of myxœdema is the administration of preparations of the thyroid gland. The success of this method is one of the most brilliant triumphs of modern experimental medicine. The results of treatment for several months are remarkable, as the entire appearance of the child is changed. The thickening of the skin disappears, the idiotic expression is lost, and the growth and development of both body and mind progress rapidly. A large number of cases have been reported illustrating the effects of the thyroid treatment. The question whether the improvement will continue indefinitely and go on to complete and permanent recovery is one for the future to decide.

The dried powdered gland or the glycerin extract are the most convenient means of administration.

The dried thyroid extract may be given in tablet form in doses of 0.03 to 0.06 gramme (½ to 1 grain) three times daily. Unpleasant effects from the treatment are rare. A decided rise in the temperature indicates that the use of the thyroid extract should be suspended for a time. The earlier the treatment is begun the better are the results obtained. When the normal condition of the body has been restored by the use of fairly large doses of the thyroid extract, it will be necessary to continue the treatment indefinitely with doses small enough to maintain the equilibrium of the metabolism of the body. It should be remembered that these children are especially susceptible to cold, and should be kept in an even temperature or taken to a warm climate during the winter.

Fig. 187 represents a case of sporadic cretinism.

FIG. 187.



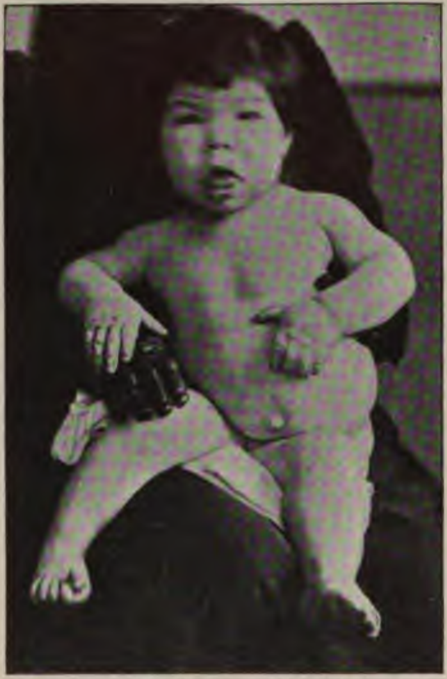
Myxœdema. Female, 5½ years old.

The child was a girl, five and a half years old. Her parents were healthy Americans, not blood relations, and did not have goitre. She was born after a severe labor; it was a head presentation, and no instruments were used. Nothing especially abnormal was noticed about her until the twelfth month, when she did not seem so bright as is usual at that age. When four years old she was brought to the hospital. She could not speak, and her mental condition was much enfeebled. She could scarcely stand, and looked as though she were about one and a half years old. She had never had any convulsions, but had always had incontinence of urine and of fæces. When seen a year later she appeared to be in good general condition, but her muscles were large and flabby and she had not improved mentally. The circumference of her head was 46.05 cm. (18¼ inches). The measurement from the occiput to the root of the nose was 34.4 cm. (13½ inches), and across the head from external meatus to external meatus 29.3 cm. (11½ inches). The circumference of the thorax was 40.3 cm. (15¾ inches). There were no irregularities about her head. The forehead was overhanging, and this was rendered more striking on account of the sunken bridge of the nose. The lips were thick, and the tongue, which seemed enlarged, was protruded between them. The hearing was said to be good, and the sight was good. She had been able to sit alone since she was one year old, but could stand only with support, and could not walk. She was bow-legged, and the bones were somewhat enlarged about the epiphyses. The hands and feet were large and puffy, but did not pit. The feet were bright red, the hands less so. The trunk was stout and thick; the spine was straight; the lungs and heart were normal, and nothing abnormal could be detected about the abdomen except an umbilical hernia. The tendon reflexes were normal. Sensation was normal. The thyroid gland could not be felt. The teeth were good. There was a general condition of infiltration of the skin like myxœdema. Hebetude was marked. The treatment of this child was with an extract made from the thyroid gland of a sheep, 0.06 c.c. (1 minim) of the thyroid extract being given three times daily, which was gradually increased 1 minim every two days unless the rectal temperature rose above 37.7° C. (100° F.). There was a slight improvement of the symptoms before the child left the hospital, but her parents did not return with her to report the subsequent progress of the disease.

Fig. 188 represents a case of sporadic cretinism which occurred in the practice of Northrup, of New York, who describes it as follows:

"The parents of the child were healthy Americans from Western Pennsylvania, and they were not consanguineous. The father was forty-five years old ; the mother was thirty-nine years of age, had had several miscarriages and four healthy children, two of whom had died of some acute disease. This little girl, who is now nine years old, is the fifth child. The mother first noticed that the child could not sit up when it was nine months old, that it practically ceased to grow, and now at nine years it is mentally no older than it was at nine months, and physically it has merely thickened. The first impression one gets on looking at the child is that it is an idiot. Its hands are large and broad. Its color is peculiarly sallow. The hair is thin, long, dry, and without lustre. The eyebrows are present, and are not remarkable in any way. She has the characteristic flattening of the bridge of the nose, diffuse swelling of the under lid and puffiness of the upper lid, and pendulous cheeks. She has thick, pale lips, with a protruding tongue, which is swollen and pale. The lips and tongue have a tendency to dryness. There are fourteen teeth ; all of them are of the first set. Those in the upper row are eroded, and appear only at the bottom of a series of ulcers in the upper gums. The lower teeth are in nearly the same condition, and the gums are suppurating. An offensive odor is always present in the mouth. The arms, legs, feet, and hands are unnaturally thick. The abdomen is prominent, and there is, as you see, an umbilical hernia. The hand which is resting on its mother's black glove shows the dry, wrinkled condition so characteristic of myxœdema. Perspiration is absent. The skin is pale, and has a peculiar mottled appearance. The soles of the feet and the palms of the hands are dry. There is marked lordosis. The surface of the child does not suggest the feeling of œdema, nor does it pit. The feeling is that of puffiness and flabbiness. The child cannot sit alone. It can, however, stand when once balanced and allowed to grasp some fixed object. The supraclavicular 'pad' of tissue so commonly found in these cases is present. The thyroid gland seems to be present, and is possibly enlarged. Hebetude is shown to a marked degree, and the delayed cerebration is very evident, although the child never speaks except to say, with infinite slowness, 'da—da.'

FIG. 188.



Myxœdema. Female, 9 years old. Slight improvement after eighty days' treatment with thyroid extract.

"The rectal temperature four days before treatment was begun was 36.4° C. (97.5° F.) in the morning, and 37.5° C. (99.5° F.) in the evening. The child was treated with the thyroid extract prepared so that each drachm represented one thyroid gland of a yearling. Of this preparation 0.06 c.c. (1 minim) was given three times a day until the fourth day, when the temperature rose above 37.7° C. (100° F.), and the treatment was stopped for a day. At this time the appetite had improved, and the breath was not so offensive. Two days later the treatment was begun again, and on the eighth day the tongue was found to be considerably smaller. During the next week the temperature remained under 37.7° C. (100° F.). It then rose above 37.7° C. (100° F.), and the treatment was suspended. The first tooth, a canine, was cut at this time. The largest dose which was given during the treatment was 0.24 c.c. (4 minims) three times a day. The child was treated eighty days in this way. The improvement was very slight, but the countenance was brighter, the tongue became much smaller, and the skin less dry. She lost somewhat in weight while under treatment. The constipation, which was marked when the treatment was begun, disappeared, and she was willing to take a much greater variety of food."

The next case represents one of sporadic cretinism which came under the observation of Osler, of Baltimore, when she was four years old.

The parents were healthy, and there was no hereditary taint on either side of the family, none of whom had had goitre. She was the second child ; the labor was easy, and she throve well. She had never had any diseases. Nothing especial was noticed about the child until its second year, when it was observed that she did not attempt to walk or talk, and that she seemed unnaturally quiet and dull. She did not cut her first teeth until she was two years old. In her third year her skin became very pale and waxy, and her face and limbs seemed puffy and swollen. She had developed very little mentally, and could say only one or two words. The other symptoms indicative of a disturbance of the function of the thyroid gland gradually appeared, such as the myxœdematous condition of the subcutaneous tissues and the development of the supraclavicular pad. The thyroid gland could not be felt. The examination of the blood showed a moderate increase of leucocytes and some irregularity in the size of the erythrocytes. When three and a half years old she was 75 cm. (29⅜ inches) tall, and her head measured 52.3 cm. (20½ inches). She had been under treatment with tonics for a year, and was reported to take more notice and to look more intelligent. She was then treated with the thyroid extract, and improved markedly in both her mental and her physical condition. The tongue, which had been thick and protruding, rapidly became smaller, and she began to walk and talk.

EXOPHTHALMIC GOITRE.

Exophthalmic goitre occasionally, but rarely, occurs in childhood, independent of the physiological disturbance which has been described under hyperæmia of the thyroid gland on page 900.

ETIOLOGY.—According to Sachs, heredity plays a much more important part in children in the production of the disease than the emotional excitement, fright, cardiac disease, and severe constitutional disorders which so frequently underlie the condition in adults. Epilepsy, chorea, and chronic alcoholism in the parents predispose to the development of the disease in children.

Exophthalmic goitre is considered by some writers to be due to a pure neurosis, by others to central lesions in the medulla oblongata. The more recent views attribute it to hypersecretion of the thyroid gland, to which the name *hyperthyrea* is given, as opposed to the *athyrea* of myxœdema, in which the symptoms are supposed to be due to deficient secretion.

SYMPTOMS.—In some cases the onset is acute, in others subacute or chronic, and is characterized by the cardinal symptoms of tachycardia, exophthalmos, muscular tremors, enlargement of the thyroid, and by general symptoms of anæmia, indigestion with a special tendency to profuse diarrhœa, slight fever, loss of weight, and many other symptoms of a neurasthenic character.

Tachycardia.—The pulse-rate is increased to 90, 100, 120, or even 200 per minute, the rate being increased in states of excitement and lowered when mental and physical rest is enforced. There is no evidence of an organic lesion in the heart. All the arteries throughout the body pulsate with unusual distinctness. Owing to the increased blood-tension, hemorrhages may occur from the nose, stomach, or intestines. Upon palpation of the goitre a distinct thrill may be felt.

Exophthalmos.—The protrusion of the eyeball is not accompanied by disturbance in vision. Limitation of the field of vision with ulceration of the cornea from want of protection of the lids may occur. Graefe's symptom, a failure of the upper lid to follow promptly a downward movement of the bulb, Stellwag's symptoms of a dilated palpebral fissure, and Moebius's symptom of defective convergence of the axes of the two eyes are signs to be noted in connection with the exophthalmos, but are not always present.

Thyroid Enlargement.—The thyroid is almost always enlarged, but the increased size may follow rather than precede the other cardinal symptoms. The enlargement is usually bilateral and symmetrical, the tissues are vascular, hyperplastic, and may show fibrinous degeneration.

Muscular Tremors.—The muscular tremors are usually rhythmical, and at the rate of about eight to the second.

Throbbing of the blood-vessels and free perspiration are unpleasant symptoms, adding to the discomfort and nervousness of the patient. Intense pigmentation of the skin, resembling Addison's disease, may rarely occur, or areas of leucoderma, or of urticaria. Very rarely myxœdema has developed towards the end of the disease. Marked diminution in the electrical resistance is present, and may be due to the profuse sweating. Glycosuria and albuminuria are occasional complications.

DIAGNOSIS.—The diagnosis is made by the recognition of the cardinal symptoms. Physiological hyperæmia of the thyroid at puberty should not be diagnosticated as exophthalmic goitre.

PROGNOSIS.—The course is generally chronic and of several years' duration. Some cases recover, but if the disease is once well developed it is apt to be prolonged, death often ensuing from some intercurrent affection.

TREATMENT.—Absolute rest in bed, avoidance of all excitement, and careful regulation of the diet are essential. Digitalis and strophanthus are sometimes useful. Good results have been reported from the use of belladonna and of atropine. The galvanic current is also recommended. The thyroid extract has proved of no value in these cases. When the gland is so large as to cause symptoms of pressure, partial extirpation is to be considered. The operation has been done in adults with good results in a certain number of cases.

ACUTE THYROIDITIS.

Acute inflammation of the thyroid is not very common, but may occur from a variety of causes. It may result in the formation of abscesses of various sizes or in the production of new connective tissue. Acute thyroiditis is rarely primary, being commonly a metastatic affection occurring in the course of some febrile disorder. It has been noticed among children as a complication of measles, typhoid fever, diphtheria, and parotitis, and the process in a majority of these recorded cases, instead of retrograding spontaneously as it did in others, caused an inflammatory condition in which abscess-formation occurred. On opening the abscesses the pus was found to contain numerous micrococci.

SYMPTOMS.—The symptoms of acute thyroiditis are swelling, redness, and tenderness of the gland. Symptoms of pressure, such as dyspnœa, hoarseness, painful deglutition, and neuralgic pains, are sometimes present with fever and general malaise. The duration of the disease is from two to three weeks.

TREATMENT.—The treatment is essentially expectant, but some previously intractable cases seem to have been benefited by the application of iodine. The patient should be carefully watched, and, if there are indications that suppuration has taken place, an incision should be made at once, as recovery then usually occurs quite quickly.

TUMORS OF THE THYROID GLAND.

Malignant growths are extremely rare in the thyroid. Gummata have been found in a certain number of cases of congenital syphilis and miliary tubercles in connection with general miliary tuberculosis.

DISEASES OF THE THYMUS GLAND.

The thymus gland may persist after the sixteenth, and even after the twentieth year, without especial pathological significance.

A considerable number of cases of what is termed *thymic asthma* has been reported.

In this condition infants and young children, many of whom were supposed to be healthy, have had severe and often rapidly fatal attacks of dyspnœa, which at the autopsy have been found to be dependent upon great enlargement of the thymus gland. Death in these cases is supposed to have been due to pressure of the gland upon the trachea, upon the great vessels, or upon the pneumogastric nerve, causing spasm of the glottis. Many of these cases have occurred in children with rhachitis.

Minute multiple *hemorrhages* are not infrequently found in the thymus gland in new-born infants who have died of asphyxia.

Primary *inflammation* of the thymus gland has been reported, but is extremely rare.

Multiple abscesses of the thymus gland have been reported by Dubois portion to the degree of anæmia present, and a characteristic discoloration of the skin are the conspicuous symptoms of the disease.

The pigmentation varies from a light yellow to a deep bronze. It is usually diffuse, and is most intense on the exposed parts of the body, such as the head and hands, and in the flexures, around the nipples and about the genitals. The mucous membranes of the mouth and vagina are likewise pigmented, but the palms and soles remain free for a long time. Small areas of leucoderma may appear.

Vomiting and diarrhœa are especially common when the disease occurs in children. Nervous symptoms are sometimes marked, and are of the same character as those we see in cases of severe secondary anæmia. The blood, however, shows only a slight degree of anæmia. There is no emaciation except in the later stages.

The course of the disease is progressive, with occasional remissions, the duration varying from a few months to one or two years. Death occurs from exhaustion or from the development of an intercurrent affection, which may appear as some form of tubercular disease in other parts of the body.

On autopsy the adrenal glands are frequently found to be tubercular, but may present no changes.

DIAGNOSIS.—In the diagnosis of Addison's disease other causes of pigmentation of the skin must be excluded, such as may arise from arsenic, lead, nitrate of silver, paludism, and abdominal growths. The tuberculin test is often of value as an aid in the diagnosis when the cause is due to tuberculosis of the adrenal glands.

PROGNOSIS.—The disease is nearly always fatal. A few recoveries have been reported.

TREATMENT.—The adrenal glands of the sheep have of late been used frequently in the treatment, and in a few cases with improvement in the symptoms. The remedy is worthy of a trial, but the results are usually very disappointing. The glands may be given raw or slightly cooked, in the form of a glycerin extract, or as a dry extract in tablets. From one-half to one gland may be given daily, or one-grain doses of the dried extract three times a day. General treatment and measures directed to the control of the symptoms are indicated.

DIVISION XVI.

DISEASES OF THE NERVOUS SYSTEM.

WE are much more likely to meet with nervous phenomena of the most diverse varieties in children than in adults. In like manner we meet with the most widely differing clinical symptoms. Symptoms which if occurring in adults would be significant of serious lesions of the nervous system may arise in children from simple reflex conditions which only simulate and do not represent actual disease.

Children are much more apt to become unconscious, to have convulsive attacks, and to show disturbance of the functions of important nervous centres from reflex irritation, than are adults. The whole cerebro-spinal system in infancy and early childhood is so impressionable, so excitable, and so hypersensitive to even slight grades of irritation, that diseases of a nervous type, whether primary or secondary, dominate all others.

Reflex phenomena are so much more numerous than those which arise from organic lesions, and are so irregular in their manifestations, that, from a diagnostic point of view, they are most important. They also enter into all disturbances of the nervous system, whether functional or organic, to such a degree that what we have learned concerning cerebral localization in the adult becomes of much less value in the young subject.

The reader is referred to especial works on the nervous diseases of children for the details of examination and cerebral localization, and I can recommend particularly the "Nervous Diseases of Children," by Sachs.

Instability and irritability of the nervous system, both peripheral and central, are characteristic of the early periods of development, making certain nervous diseases of children peculiar to them as compared with adults. Many weeks are required before the peripheral nerves have acquired their complete function, and the brain and spinal cord do not attain their full development for months and years. A simple heightened temperature or increase of vascular pressure in the brain and cord may cause such irritation of the nervous centres that the most varied symptoms, such as delirium, somnolence, and twitching, may arise. On the other hand, serious nervous disturbances may in childhood follow the acute infectious diseases, and again there may be a complete arrest of development of the central nervous system, which may be temporary or permanent.

As stated by Sachs, childhood is exempt only from the diseases due to senile deterioration from degeneration and sclerosis of the brain and spinal cord, and is relatively free from those due to toxic agents, such as alcohol, metallic poisons, and acquired syphilis; the effects, however, of such diseases in the parents are shown by inheritance in the children.

CONVULSIONS.—ECLAMPSIA INFANTUM.

Attacks of motor disturbance represented by continuous rigidity or contractions of one or more groups of muscles, lasting for a variable time and usually accompanied by unconsciousness, are designated convulsions. A convulsion is a symptom and not a disease.

Convulsions may be divided, as to their type, into (1) *clonic* and (2) *tonic;* as to their form, into (1) *general* and (2) *partial;* and as to the seat of irritation which causes them, into (1) *central* and (2) *peripheral.*

The *clonic* convulsion is an active spasmodic contraction of the muscles followed by immediate relaxation. The convulsions of epilepsy are illustrative of this type.

The *tonic* convulsion is a more or less continued spasmodic rigidity of the muscles. This type is seen in tetanus neonatorum.

The seat of irritation which produces the convulsion is very varied. It may be a lesion of the central nervous system or of the peripheral nerves; in the former case the convulsions are spoken of as *central*, in the latter they are termed *reflex.* Convulsions are much more apt to occur in infancy than in later childhood and in adult life, probably because the power of inhibition is not developed in the former. We therefore not only meet with convulsive attacks more frequently in very early life, but, as a rule, we are led to look upon these convulsive attacks as of much less import than in the older subject. The reason for this is that the causes of reflex convulsions in infancy are innumerable, and, as a rule, do not result seriously, while in older children and in adults convulsions are almost always representative of some serious central lesion. Convulsions are so common in infancy that they have been compared to the chill which occurs in the initial stage of many diseases arising in adults. The various acute diseases accompanied by high temperature, such as pneumonia and the exanthemata, are very commonly preceded by a convulsion, and a chill is rare under these conditions in infancy. We must, however, not be misled by the frequency and comparatively benign character of convulsions in infancy and by the rule that they are not fatal. The convulsions of infancy may represent just as serious conditions as they do in later life, and are to be looked upon as a grave symptom until we can be sure, by eliminating serious organic lesions as a cause, that we are dealing with one of the common and mild forms of this phenomenon. The convulsion does not in itself show us whether it is the result of serious or of benign disease, and the convulsions which occur from some organic lesion, such as cerebro-spinal meningitis, may differ in no way from those which arise from some simple cause, such as indigestible food.

General Symptoms.—We are frequently called to see infants in convulsions where the convulsion is the first and only manifestation of the disease. After a few signs of uneasiness the infant suddenly becomes rigid for a second or two, makes a sound as though choking, the eyes turn upward and become fixed, there may be strabismus, the skin becomes somewhat cyanotic, and then the convulsive movements begin. The eyelids open and shut; the face and usually the head are drawn to one side; the fingers are clinched, and the arms and legs move up and down. The back may at times be somewhat arched and the head retracted. The infant is apt to foam at the mouth to a greater or less extent; it is perfectly unconscious, and the breathing soon becomes stertorous. These symptoms, after lasting for two or three minutes, are followed by complete relaxation, an apparent state of coma, and sleep. The child on awakening may be bright and well, or the convulsion may recur and continue for a much longer time, as in one of my cases, in which an infant had fifty-two convulsions in twenty-four hours and yet recovered. There may be involuntary discharges of urine and fæces.

The convulsive movements may affect the entire body and limbs, including the face, or they may affect only certain groups of muscles. Thus, they may be localized, as in one limb, or they may be unilateral or bilateral.

Convulsions of Central Origin—The most important, on account of their serious nature, are those convulsions which are of central origin. Convulsions of this nature may occur in any disease which is represented by a high temperature, such as insolation, meningitis, the exanthemata, pneumonia, or other diseases. In these cases the convulsions are produced either by the action of the high temperature on the motor centres of the brain, or by the direct action of the special toxic agent which is producing the disease. These convulsions, as a rule, are general, and are produced by the diffuse action of the poison. In this class of cases it is probable that there is an extremely hyperæmic condition of the blood-vessels of the central nervous system. The convulsions may also, in contradistinction to the supposed theory of active hyperæmia of the blood-vessels and the high temperature, be produced by vascular stasis and a normal or subnormal temperature. This form may occur in the course of pertussis or of cardiac disease. Again, central convulsions are supposed to be caused by an anæmic condition of the blood-vessels of the brain, such as may arise from loss of blood or from exhausting diarrhœa. Such toxic agents as are represented by drugs of various kinds, as belladonna, may produce general clonic convulsions. Mental disturbance, such as sudden fright, has also been known to produce a convulsive attack. In all these classes of cases the convulsions may be partial and clonic instead of general, although, as a rule, they are general, owing to the diffuse character of the irritation. In addition to these convulsions which arise from a diffuse cause are others in which a local lesion having occurred in the brain from morbid growths, embolism, thrombosis, hemorrhage, or any other cause, a disorganization of a portion of the brain has resulted. As these lesions are generally focal in their distribution, the convulsions are apt to be localized and hemiplegic in character as in cerebral paralysis.

A number of diseases can by their direct effects, irrespective of the temperature which accompanies them, produce convulsions. Thus, convulsions occur not uncommonly in the course of nephritis, in which they are usually called uræmic, also in malaria and in other diseases. Direct pressure from tumors of the brain or from hydrocephalus may in like manner cause convulsions of either a localized or a general form. Finally, we may have these nervous explosions in epilepsy.

It will be well to remember that this entire class of central convulsions emanates from the brain ; and also that when the convulsions are unilateral or localized we should suspect a central rather than a peripheral origin.

Convulsions of Peripheral or Reflex Origin.—The other class of convulsions spoken of as of peripheral origin, and which are called reflex, have so many causes that it would scarcely be advisable to attempt to enumerate them all. Convulsions of this class may arise from almost any source in infants whose nervous system is so easily irritated that the slightest cause may produce a nervous explosion. The disease which most commonly gives rise to convulsions of the reflex form is rhachitis. Rhachitic children seem to be predisposed to spasmodic attacks of all kinds, and a general clonic convulsion in children with rhachitis corresponds to the spasmodic contractions in the larynx which occur in rhachitis, and which is spoken of as laryngospasmus.

It is probable that there is no special lesion, in connection with the rhachitis, which necessarily gives rise to convulsions, but that all the tissues in this disease are especially sensitive to causes which may produce reflex explosions.

The most common cause of reflex convulsions in infants is improper food. Convulsions from this cause arise not only when manifestly indigestible articles are given to young children, but even in infants who are being fed from the breast. In the early days and weeks of life, before the breast has acquired its normal functions connected with the elaboration of milk in which the solids are in proper proportion to each other and to the water, it is not uncommon for the infant to have convulsions produced by a disturbance of the mammary function. In cases of this kind it is usually found that the percentage of the proteids is high, and that the convulsions will continue until this high percentage has been lessened, if the infant is allowed to continue to nurse. Whether the teeth of themselves during their development are a source of sufficient irritation to produce convulsions has been questioned by many observers. It is, however, evident that during the different periods of dentition reflex convulsions are more apt to occur than when a tooth is not disturbing the infant's nervous system. In addition to the convulsions arising from improper food in the stomach during the dental period, foreign bodies in the intestine, whether in the shape of food or in that of intestinal parasites, may cause reflex convulsions. Foreign bodies in the nose and in the ear have been known to produce convulsions, as has also an inflamed tonsil in the initial stage of a follicular tonsillitis. Hot baths are so often given to infants when they are in convulsions that they should be spoken of as a source of convulsions, for they have been known to produce this result when care has not been taken to test the temperature of the bath before the infant is put into it. Mental disturbances, such as arise from fright and other causes, are also etiological factors in reflex convulsions.

PROGNOSIS IN CONVULSIONS.—The prognosis in infantile convulsions varies with the especial cause. On recovering from the attack the infant may show signs of some serious central lesion, such as paralysis, or may be left apparently perfectly well. A single convulsion followed by perfect recovery is of slight consequence, but when the convulsive attacks recur frequently and last longer than in the attacks which have just been mentioned, the prognosis becomes more grave. Even though no central lesion be present, continued convulsions may, by the shock to the infant's vitality, finally cause death from exhaustion; or death may occur from spasm of the glottis. Numerous convulsions, however, do not necessarily lead to a fatal issue. We must, therefore, irrespective of the cause or the apparent result of a convulsion, always look upon it as a grave symptom and endeavor to prevent its recurrence.

TREATMENT.—When summoned to treat an infant who is in convulsions, we should first see that the bath, in which we usually find that it has been immersed, is not too hot, and should order the infant to be taken out of the bath before it becomes conscious, or it may be so frightened as to excite again the reflex spasm. The thorax should be quickly examined for pulmonary and cardiac lesions, and inquiries should be made as to the history of the case, with special reference to the infant's diet. The temperature should be taken, and the fontanelle be examined for bulging or depression.

Having obtained this information, we can eliminate a number of causes for the attack, such as the onset of one of the exanthemata if the temperature is normal, and reflex convulsions from food or from foreign bodies in the nose or the ear. If the convulsions arise from exhaustion, stimulants should be given at once, and if the convulsions continue, the general treatment which is indicated for all forms of convulsions is indicated. The treatment should be directed to the especial cause of the convulsion, if any can be determined. As, however, it is often impossible when the convulsion is present to determine whether it is of central or of peripheral origin, it becomes necessary to endeavor to control the attack at once by general treatment. For this purpose the inhalation of ether in small amounts, and the emptying of the bowels by means of enemata, are indicated. When the convulsions are of a very severe type, continuing with perhaps intermissions of only a few minutes, and the infant's life is evidently in danger from the continuous shocks which are taking place to its nervous system, a rectal injection of from 0.18 to 0.48 gramme (3 to 8 grains) of bromide of potassium and from 0.06 to 0.30 gramme (1 to 5 grains) of hydrate of chloral in 30 c.c. (1 ounce) of warm water, repeated if necessary every hour for three or four doses, is indicated. If the convulsions still continue and a fatal issue is anticipated, a subcutaneous injection of sulphate of morphine, beginning with 0.001 gramme ($\frac{1}{60}$ grain), should be tried.

In most cases of infantile convulsions, of whatever form, the warm bath at a temperature of not over 37.7° C. (100° F.) can be used, for, although it is not in any sense curative, it tends to quiet the nervous excitability and to lessen the muscular strain produced by the continuous spasmodic muscular contractions. The class of cases in which this is contraindicated are those which are caused by a loss of blood, an anæmic condition, diarrhœa, and cardiac disease, and those in which venous stasis is present with a lowered temperature. In these cases stimulants are indicated.

When there are symptoms of the diseases which I have already spoken of which are accompanied by high temperature, the reduction of the temperature by the bath and the administration of an antipyretic are indicated.

The treatment of convulsions caused by definite diseases is simply symptomatic while the convulsions continue, and later appropriate treatment of the primary condition.

The following cases represent eclamptic attacks from various causes:

A little girl, six and one-half years old, was healthy at birth, and had never had any disease. When three and one-half years old she had from time to time convulsions, clonic in type. When in the convulsions she did not bite her tongue. The first convulsion occurred when she was three years old; the next when she was four years old; the next when she was four and one-half years old; and the last one when she was five years old.

As all these convulsions were apparently produced by the same cause, it will only be necessary to describe them in a general way. They were characterized by sometimes continuing much longer than is usual in infantile convulsions, one of them having lasted for one hour and a half, during which time the hands were clinched, the eyes were rolled up, and the entire body and limbs were convulsed. Previous to each attack the child for a number of days had indefinite symptoms, which she could not describe accurately, connected with the abdomen and accompanied by a feeling of weakness and slight muscular twitching.

At the time of the earlier attacks her mother found that these symptoms could be dissipated and a convulsion apparently prevented by giving her a dose of castor oil about once a week. After the third convulsion she passed a lumbricoid worm. From that time whenever she showed the premonitory symptoms of a convulsion she was treated with santonin followed by a cathartic, a lumbricoid worm each time was passed, and the symptoms disappeared. When she was five years old she was thoroughly treated for these lumbricoid worms with santonin, and after a large ascaris had been passed the nervous symptoms ceased entirely. The child when last seen had not had a convulsion for over a year. There had been no reappearance of the parasites.

This child represents the class of cases described as reflex convulsions, the cause of the peripheral irritation being an intestinal parasite.

Another case was that of an infant, four months old. Her mother, who was strong and well and apparently had plenty of good breast-milk, nursed her at birth. When she was three months old she began to have convulsions, which occurred almost every hour. Suspecting that the proportion of solids in the breast-milk was too high for the infant to digest, and that it was producing a peripheral irritation which was the cause of the reflex convulsions, I had an analysis of the milk made, and found that the proteids showed a percentage of from 4 to 5. The infant was then fed with a carefully modified milk in which the percentage of the proteids was made 1. Within a few hours the convulsions ceased, and they have never returned.

A case similar to the one just described was that of a boy three weeks old, previously healthy and strong, and digesting his mother's milk perfectly; he was weaned and given by mistake a mixture containing a total proteid of 3.50. He then began to have convulsions, which continued until a new mixture was given, in which the total proteid was 1.50, when they ceased.

CHOREA. SYDENHAM'S CHOREA.

Chorea is a disease characterized by irregular and involuntary muscular movements without loss of consciousness, and affecting the muscles of volition.

The disease is rare in infancy, but may occur in the early months of life. It seldom begins after puberty. It is most apt to begin and is most marked in its symptoms during the period of the second dentition,—that is, during the period of active growth, from six years to puberty. The greatest number of cases is found among the female sex and among those who do not receive sufficiently nutritious food. A sharp distinction should be made between the disease chorea, with its characteristic choreiform symptoms, and the same choreiform symptoms resulting from other diseases, sometimes represented by central nervous lesions, sometimes by purely reflex causes. It will save much useless reading of the literature of chorea and much profitless discussion as to its etiology and pathology if this distinction is borne in mind. Eliminating those forms of chorea which are due to gross lesions of the nervous system, such as the post-hemiplegic and congenital forms, we can at once very materially reduce the cases of true chorea. In like manner we should separate from true chorea those cases of peripheral irritation in which the partial choreiform symptoms are evidently reflex and can be cured by removal of the cause. Examples of these reflex choreiform symptoms are the facial chorea from naso-pharyngeal irritation and the partial choreiform movements occasionally arising from errors of refraction and ocular insufficiency. The consideration of these anomalous forms of chorea belongs with the diseases in which they occur.

Etiology.—Chorea can be brought about by other diseases, such as measles, although this, in my experience, rarely occurs, and even then chiefly among the poorly cared-for. A certain number of cases have so directly followed intense fright that we must acknowledge acute mental conditions as a cause. The disease which is most frequently associated with chorea is rheumatism. The percentage of cases, however, in which this association takes place is difficult to determine. This difficulty arises from the want of uniformity in the reported cases of different observers, due to their different ideas as to what constitutes rheumatism. If only the cases of acute articular rheumatism are to be classified under rheumatism, very few cases of associated chorea need be spoken of; while if all the flitting aches and pains of childhood are considered to be rheumatism, the number of choreic cases caused by rheumatism rises to fifty per cent., or possibly more. The truth will in the future probably be found to lie in some intermediate number, for in certain cases a close connection exists between chorea and rheumatism. The difficulty becomes still greater when we examine the relationship between chorea and endocarditis. When there is a rheumatic element in the case we should expect a cardiac lesion to arise which may be dependent on the rheumatism. In certain cases, however, we find chorea with endocarditis entirely irrespective of rheumatism. This occurs to such an extent that in chorea we should watch for cardiac lesions just as carefully as in acute articular rheumatism. Heart-murmurs of a hæmic nature may occur in chorea as in any other disease of a debilitating nature. They should, however, always be looked upon seriously, as possibly indicating an insidious form of organic endocarditis, which, instead of being evanescent and passing off entirely with the recovery of the chorea, may either seriously disable the heart or lead to a fatal issue. A special microbic cause for chorea, as for rheumatism, must be thought of, but as yet has not been proved to exist. An hereditary tendency to nervous explosions of a choreic type has long held a prominent place in the etiology of chorea. This tendency does not manifest itself, however, unless the children are poorly nourished, badly cared for, or exposed to nervous excitement during their school life.

Overtaxing of the central nervous system during the school year has so often been shown to result in an attack of chorea in the spring and in a recurrence in the autumn on returning to school, that it should be recognized in considering the etiology of the disease. Strain of the ocular muscles has been considered an exciting cause of chorea, but beyond causing in some cases choreiform symptoms it cannot be considered as being an inciting cause of true chorea.

Pathology.—There are a large number of cases of chorea in which the disease is found to have no apparent pathological lesion. Its symptoms, however, show us that the morbid process is located in some part of the central nervous system. Whatever the nature of the lesion, it is represented by a profound excitement of the motor centres, presumably due to their inanition, and is accompanied by a temporary inability of these centres to recover themselves. Many lesions have been described as occurring in chorea, but in the pure cases (Sydenham's chorea) which have just been described, and which really represent the disease, there is no lesion which with our present knowledge we can say is characteristic.

SYMPTOMS.—Chorea may be in its distribution general or partial; in its course acute, subacute, or chronic. In many cases the disease is exceedingly mild in its symptoms and is of a benign type; in others it assumes a severity which seems to threaten life. The beginning, although at times sudden, as from fright, is, as a rule, gradual, at first a few muscles only being affected. The child becomes fretful and impatient. Its irritability must carefully be differentiated from bad temper, for which the symptoms are apt to be mistaken by the family. The clinical picture of the disease is a jerky, irregular, involuntary contraction and relaxation of the muscles, which usually begins in the fingers, hands, and face. There is an irregular, uncertain action of the part affected, and efforts of the will only partly control the movements. As the disease progresses, the voluntary control of the muscles diminishes more and more, and at times disappears entirely.

The movements ordinarily cease during sleep, but in severe cases they continue and even interfere with it. At times the child is unable to walk, on account of weakness. The speech may become slow and indistinct, from the affection of the muscles of the tongue and of the larynx, and even mastication and deglutition may become difficult. In very severe cases the difficulty in speech may be enhanced by the mental condition, which may be represented by dulness and apathy. The tendon reflexes are lessened in severe cases. The muscles grow weak and soft, and there is considerable emaciation. There is usually loss of appetite, and the bowels are often constipated. The urine and its urea have been found to be increased during the course of the disease. The dynamometer usually shows impaired muscular power. In certain cases the muscles of the extremities on one side of the body are principally or alone affected (hemichorea). These cases do not differ from the ordinary bilateral cases in any way except in this respect.

In very severe cases there may be involuntary evacuations of the fæces and of the urine. The disease is distinct from epilepsy, and there is little danger of the patient becoming epileptic unless the disease happens to develop in an individual who is predisposed to that condition.

PROGNOSIS.—Chorea is very apt to show relapses and to recur for several years. Although often obstinate in the persistency of its symptoms, yet it may be said to be self-limited, and, as a rule, to recover, provided no complications, such as of the heart, arise. The time which elapses before complete recovery is very variable, but well-marked cases usually extend over a period of three or four months. Although chorea is considered, as a rule, a benign disease, yet we must always look upon it as a serious disturbance until we are sure that we are dealing with the usual mild form of the affection. The following case illustrates how careful we should be to give a guarded prognosis in the early stages of acute chorea:

A girl, nine years old, reported by Cook and Beale, began to have choreic movements, which constantly became worse. Delirium developed, with a slight fever, a rapid and feeble pulse, and a quick and interrupted respiration. Death suddenly occurred one hundred and thirty hours after the onset of the disease. The autopsy revealed extreme anæmia of the pons and medulla, but no other changes of note in other parts of the body.

We must allow that even uncomplicated chorea is a varying disease as to the severity of its symptoms and their persistence for a longer or a shorter time. We also know that there is a marked tendency to relapse, and that the number of relapses varies to a great degree. The length of the attack and the response to treatment may differ much. Bearing these facts in mind, we can understand the rapidity with which certain individuals are attacked or the quickness with which they recover. Some cases recover rapidly under only hygienic treatment, while others are apparently unaffected by any drug whatever. When heart-murmurs, evidently representing organic disease, appear, it is often a cause for comment at the comparatively slight discomfort which the cardiac lesions entail. At times, again, it is surprising how rapidly fatal are some cases which are complicated by cardiac disease, and how they are uncontrolled by any treatment whatsoever. The disease is variable in its duration whether treated by drugs or not.

TREATMENT.—There cannot be said to be any specific treatment with drugs for chorea, but of the many remedies that have been used, arsenic has, in my experience, been the most beneficial. It should, however, be used with care, and on the appearance of any evidence of the physiological action of the drug, such as nausea or œdema of the eyelids, it should at once be discontinued. It should not, as a rule, be given in very large doses, as cases have occurred in which it has produced a multiple neuritis of many months' duration. When any special cause can be found for the attack, such as rheumatism, appropriate treatment directed to that cause should be employed. It is manifest, however, in the uncomplicated cases that our main reliance must be placed on hygiene and food. Fresh air, nutritious food, tonics to control the anæmia and general prostration, kindness, seclusion to secure mental quiet, stimulants if there is much resulting weakness, and the bromides for insomnia and over-excitement, are the means which I have found most valuable in managing this disease. I have seen well-marked cases get well in from sixty to seventy days where good food and a small amount of stimulant constituted the entire treatment.

If the attack is very severe, skilled nursing is a very important adjunct in the treatment. The child should be protected from harming itself by means of a padded bed, and light but well-padded splints to control the movements during sleep are indicated occasionally.

The following cases illustrate the different phases of chorea:

A girl, six years old, represented one of the milder forms of chorea. There was no history of nervous or cardiac disease or rheumatism in the family, and the child herself had never been sick before.

She first complained of pain in her left hand and arm, and later the muscles of the arm began to twitch. Soon after the whole body was affected in the same way. Somewhat later it was found that the child could not talk plainly, and it was with some difficulty that she could feed herself. She seemed nervous and peevish, and showed constant irregular incoördinate movements, chiefly of the face, mouth, and upper extremities. The legs were slightly affected, and sometimes the muscles of the trunk. There had been no paralysis of the muscles. The eyes were normal in their reaction.

She was treated chiefly without drugs, and especial attention was paid to giving her a nourishing diet, baths, gentle massage, and rest in bed in a quiet room. A physical examination showed nothing abnormal in connection with the heart or other organs. An examination of the urine showed it to be normal. Marked improvement was shown after she had been treated for two weeks, and at about the forty-second day from the onset of the disease she had recovered completely.

Another girl, thirteen years old, represented one of the milder forms of recurrent chorea.

She had a number of diseases preceding her first attack of chorea. When she was two years old she had diphtheria, when five years old, measles, when seven years old, scarlet fever, and when eight years old, rheumatism. When nine years old she had her first attack of chorea, which occurred in the spring of the year, and lasted for a number of months. This was followed in the spring of the next year by a second attack. In the spring of the following year she had a third attack of the disease; at this time the incoördinate movements were not so marked as in the previous attacks, but the debility was greater. When she was examined during this attack, it was found that the heart, although weak and somewhat irregular, presented no evidence of murmurs. The pulse was 84, the temperature was normal, and there were no signs of any other disease. She was treated at the hospital, and recovered in a few months. In the spring of the next year she had a fourth attack of chorea. At that time nothing abnormal beyond the choreic movements was noticed. She was treated with from 0.18 to 0.36 gramme (3 to 6 minims) of Fowler's solution three times a day, and in a few months left the hospital apparently well.

In the spring, one year from the beginning of the fourth attack, she entered the hospital with a fifth attack. On this occasion, after using Fowler's solution for a few weeks and not obtaining any special benefit, iron and nux vomica were given. She gradually improved under this treatment. There remained for some time, however, incoördinate movements, which appeared especially when she was embarrassed by the observation of the people who were around her. It was a case in which a good prognosis could be given, as there were no cardiac or other complications.

The following case illustrates recurrent chorea with the development of a functional disturbance of the heart, resembling organic disease.

A boy, nine years old, was subject to attacks of chorea for nearly four years. The attacks usually came on in the spring with considerable severity, and continued for nearly six months, gradually diminishing in intensity until the symptoms were scarcely noticeable. The child had a history of rheumatism, not, however, of a high grade. The attack began four months previous to entering the hospital, and was quite a severe one, so that he had been unable to control the movements of his hands and face during the day; they were, however, quiet at night.

On entering the hospital a physical examination showed a marked systolic murmur, heard most distinctly at the apex and transmitted to the axilla. The area of cardiac dulness was not especially enlarged. The urine was normal, and nothing else abnormal was detected about the child.

He was at first treated with Fowler's solution, 0.12 gramme (2 minims), three times a day. After four days the choreic movements became less marked and the cardiac souffle less distinct. Two days later, however, the Fowler's solution had to be omitted, as it caused nausea and vomiting. At this time there was a double souffle, heard most distinctly over the left third interspace, close to the sternum. A few days later Fowler's solution was renewed, but as it caused gastric disturbance, again had to be omitted, and it was found that it could not be given for more than two days at a time without causing puffiness of the face. The treatment, therefore, consisted chiefly of rest in bed, good food, bathing, massage, and the administration of iron in the form of the citrate of iron and potassium.

One month later the choreic movements had decidedly lessened, and the cardiac murmurs disappeared. Two months from the time he entered the hospital he was free from all symptoms of the disease.

The next case illustrates a severe case of chorea, following an attack of influenza, and terminating fatally, without the development of any complication:

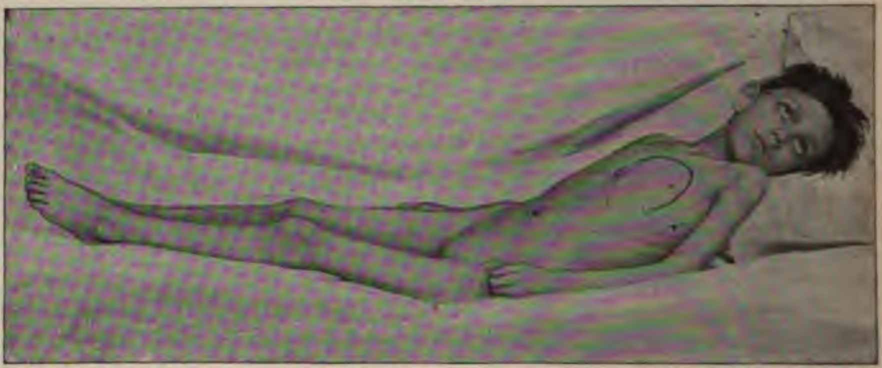
A boy, ten years old, had always been delicate, but had had no especial disease, such as rheumatism, until six weeks previous to the time when I saw him, when he was said to have had an attack of epidemic influenza. He recovered completely from the disease in ten days. Three or four days later he began to show symptoms of chorea. These symptoms gradually increased in severity, and finally were continuous, except when he was asleep. After he had had the chorea for one week he was unable to articulate, and began to have trouble with deglutition. He soon lost the control of his limbs, grew very weak, and was confined to his bed. There was considerable insomnia. In the second week of the attack the choreic movements became so violent as to endanger his falling out of bed. The temperature up to the time when I saw him, in the fourth week of the attack, was normal. The pulse varied from 140 to 150, and the respirations from 35 to 40.

When I saw him, at the end of the fourth week from the beginning of the chorea, his mind was perfectly clear; he had a little pain in the hands and shoulders, apparently from the continual movements. He was unable to articulate clearly. There was difficulty in swallowing, and he was considerably emaciated. Nothing abnormal was found in the lungs. The heart was beating tumultuously. The area of cardiac dulness was very slightly enlarged, but there were no cardiac murmurs. Although in many of these severe cases of chorea no evidence of cardiac disease can be obtained on physical examination beyond a slight dilatation of the left ventricle, yet some disease of the endocardium or valves may often be found at the autopsy. In these cases, however, the temperature is, as a rule, raised. In this case the continuous normal temperature and the absence of any signs of cardiac disease beyond a slight dilatation from the apparent weakness of the ventricular muscles seemed to indicate that it was a case of simple chorea without disease of any of the organs.

Although the child was carefully nursed and remedies of various kinds were employed to strengthen the action of the heart and to support his general strength, he failed rapidly, and died of exhaustion a few days after I saw him.

In the following case (Fig. 189) we have an example of acute articular rheumatism with endocarditis, in the course of which chorea developed with other complications, which terminated fatally:

FIG. 189.



Rheumatic arthritis. Endocarditis. Cardiac enlargement. Chorea. Female, 8 years old.

The child was eight years old. She had been subject to attacks of rheumatism. She had not had any especial diseases, with the exception of an attack of measles when she was three or four years old, until she had an attack of rheumatic arthritis when seven and one-half years old. At that time she was confined to bed with fever, and with pain, tenderness, and swelling in all her joints, especially of the knees and fingers. Although she recovered from the acute symptoms of the rheumatism, she had since then never been able to use her arms and hands, nor able to walk much. There was no record of the condition of her heart during the attack of rheumatism, but there was no history of previous cardiac disease. During the course of the rheumatism there were no other especial symptoms noticed, except that her disposition was evidently much changed and she became peevish and fretful.

One week before entering the hospital she began to have choreic movements. They were moderate in degree, but incessant. A few days later the incoördination of the muscles was also noticed when she endeavored to speak or to swallow. There were continual choreic movements of the eyes, face, and fingers, and, although seemingly she could understand what was said, she was unable to speak clearly. She was much emaciated. Incoördinate movements of all the muscles of the face, eyes, head, neck, body, and extremities were present. The peculiar look which occurs in these cases, expressive of embarrassment, was clearly shown. Although the case was a severe one, the mind was not affected beyond a slight degree of hebetude. On physical examination the lungs were found to be normal. On examining the cardiac region the impulse of the heart was found to be outside of the mammillary line and in about the sixth interspace. On palpation the contractions of the heart were clearly felt, but were irregular and not so strong as normal, suggesting irritability and incoördination of the cardiac muscles. On percussion there was normal resonance to the right of the sternum and under its upper part as far as the third right interspace. There was dulness under the sternum, beginning at the second left interspace, extending across to the third right interspace, and involving the lower part of the sternum. The area of cardiac dulness is marked in black in Fig. 189. It extended upward to the left of the sternum as far as the second rib, then to the left and downward outside of the mammillary line until it reached the impulse of the heart in the sixth left interspace. On auscultation a murmur was heard most distinctly with the first sound at the apex of the heart, and was transmitted to the axilla and to both sides of the back. This murmur was transmitted to the base, but gradually lessened as the area of the large vessels at the base of the heart was reached. Nothing else abnormal was detected on physical examination.

This was a case in which during the course of a rheumatic attack an endocarditis had developed. This endocarditis had been followed by enlargement, mostly represented by dilatation of the left ventricle. During the course of the rheumatism and of the cardiac complication the chorea had developed.

The prognosis in a case like this must be very guarded. In some instances the disease, or rather combination of diseases, grows rapidly worse, and the child dies seemingly from exhaustion. In cases of a milder form the child gradually recovers from its chorea and from its rheumatism, but is left with an organic disease of the heart from which it never recovers. The cardiac disease, however, can in most cases be much benefited by careful treatment, especially by rest in bed. In these cases the dilatation grows decidedly less, while the heart becomes stronger, and, as the chorea passes away, may show a normal area of dulness. We may at any time expect in place of gradual improvement a decided increase in the severity of the symptoms. The valvular lesion of the heart may become much more extensive, assuming the ulcerative form which is usually so fatal. The pericardium may become affected, and broncho-pneumonia occur as a complication.

The treatment in this case was with milk and stimulants. It was impossible for her to take solid food, and the milk was with the greatest difficulty introduced into her mouth.

The subsequent history of the case was as follows : The temperature rose to 40° C. (104° F.), and was accompanied by precordial pain and a pericardial friction-rub, but no physical signs of effusion. There was dulness under the left clavicle, but at first, beyond rather indefinite signs of bronchitis, nothing abnormal was detected in the lungs. The liver was somewhat enlarged. The axillary lymph-nodes were enlarged. There was great atrophy of the muscles. There was diarrhœa and incontinence of urine. The hebetude increased. Areas of dulness and diminished respiratory sound were detected. There was dyspnœa and rapid failure in strength. The child died on the thirty-seventh day from the beginning of the disease.

The autopsy showed the following lesions : Chronic pericarditis and mediastinitis ; acute ulcerative endocarditis ; thrombosis of the innominate and left jugular veins ; broncho-pneumonia ; passive congestion and œdema of the pericardium.

I have tried various methods of administering the milk and stimulants in these cases, but have found that the jaws close so spasmodically whenever a spoon is introduced between the teeth that the milk is usually spilled before it enters the mouth. The method which I have found to be most successful is by a feeding-cup with a rubber nipple fitted to the neck of the cup. The rubber nipple is perforated with a large hole.

The soft rubber does not incite the choreic movements of the jaw to the degree that anything hard would do. The Breck feeder (page 272) is also useful in these cases.

CHOREIFORM DISEASES.

Hereditary or Huntington's Chorea.—This form of chorea differs from Sydenham's chorea chiefly in its hereditary character and in the much greater degree of muscular incoördination. It almost invariably appears between the ages of thirty and forty. It need merely be mentioned in connection with children on account of its hereditary character.

Habit Chorea.—This condition is common in children. It includes simple incoördinated movements, usually of the eyes, mouth, shoulder, or thighs. They have been called "habit spasms" or "tics." Imitation is probably a strong factor in the etiology. It may follow from true chorea. These habit spasms may become chronic and persist throughout life. The treatment consists entirely of discipline of the child, and the earlier this is begun and the more strictly it is enforced the better it will be for the child.

Chorea Electrica.—This is a very rare condition occurring in childhood and adult life, and seen chiefly in Italy. It is characterized by violent spasmodic movements of the neck and head and sometimes of the extremities. In the course of a few months paralysis occurs in the affected muscles with marked atrophy and loss of faradic irritability.

Myotonia Congenita.—Myotonia congenita (*Thomsen's Disease*) is characterized by an inhibition of the voluntary movements. This disturbance of movement is due to a stiffness and tension of the muscles occurring at the beginning of motion. The most important etiological factor in the disease is that it is hereditary. In almost every case it begins in early childhood.

An examination of sections of muscle taken from cases of this disease has shown an enormous hypertrophy of all the muscular fibres, great proliferation of the nuclei, and a slight increase of the perimysium. The disease appears to be a congenital affection of the muscular fibres.

SYMPTOMS.—The symptoms are noticed only during voluntary movements, the contraction of the muscles responding very slowly to the will, and persisting for a little time after the individual has willed the muscular movement to cease. After a period of quiescence, the muscles become stiff and respond to the will only with difficulty. A tap upon the muscles causes a slow, tonic contraction, which relaxes slowly. Long-continued rest, heat, cold, and excitement cause an exaggeration of the symptoms. The muscles of the arms and legs are those usually implicated. Sensation and the reflexes are normal. The muscles are apparently enlarged, giving at times the appearance of hypertrophy, but the strength of the muscle is not proportionate to its size. Erb has described a characteristic electrical reaction, called the *myotonic reaction*, in which the contractions caused by either current attain their maximum slowly and relax slowly, and wave-like contractions pass from the cathode to the anode.

Diagnosis.—The diagnosis is made by the peculiar contraction which follows a slight tap on the muscles and by the presence of the myotonic reaction.

Treatment.—Although at times it may recover temporarily, the disease is incurable, and there is no known treatment which is of much benefit, although active exercise is indicated and is in most cases desirable.

Congenital Paramyotonia.—Congenital paramyotonia is a rare condition sometimes resembling myotonia congenita. It is an hereditary affection characterized by tonic spasms lasting from several minutes to several hours and coming on soon after birth. There is no myotonic reaction.

Paramyoclonus Multiplex (*myoclonia*).—The principal symptom of this condition is a clonic spasm of the muscles of the extremities, the face generally being exempt. The condition is very rare in childhood. The attacks occur at intervals. Symmetrical muscles are affected, and there is but slight myotatic irritability.

EPILEPSY.

Epilepsy is presumably an organic disease of the nervous system in which the pathological lesion has not yet been determined.

The characteristic symptoms are attacks of unconsciousness with or without convulsions, with a great liability to a recurrence of these attacks through a long period of time. The transient loss of consciousness without convulsions which occurs in epilepsy is called *petit mal*, while the loss of consciousness with general convulsive manifestations is called *grand mal*. Convulsions precisely similar to those occurring in true epilepsy may occur in organic cerebral disease as the result of external traumatism or from other causes; such convulsions have been termed *epileptiform*. The term *Jacksonian epilepsy* is applied to localized convulsions which are the result of organic cerebral affections. These latter forms must not be confounded with true epilepsy.

It is important that a sharp distinction should be made between the convulsions of true epilepsy and the many reflex convulsive attacks which come from a variety of causes and arise from the hypersensitive condition of the infant's nervous system. These reflex convulsions so closely resemble the convulsions which occur in epilepsy that the great importance of distinguishing between the two diseases can hardly be exaggerated. In the infant's rapidly growing brain the irritability of certain motor centres is physiologically far greater than in later childhood and in adult life. This irritability is the source of nervous explosions produced by many causes, often slight in their nature, and it is impossible to differentiate these explosions by their clinical symptoms alone from the convulsive attacks of epilepsy.

Etiology.—It is usually granted that the initial lesion of true epilepsy lies somewhere in the cortical motor centres of the brain, and that the epileptiform convulsion is an irritation of these centres. True epilepsy may, of course, originate in early infancy, and does so in a large number of cases. Whether, however, infantile convulsions may be the cause of epilepsy is a very different question. The fact is that we do not as yet know what produces epilepsy. The various etiological factors which are usually cited, such as fright, injury, and dentition, probably have nothing more to do with the production of the disease than to precipitate its development in an individual who is already predisposed to it. Inheritance as a cause of epilepsy will presumably, in the future, hold a much less prominent place than has been granted to it in the past.

There is no good reason for believing that reflex convulsions in themselves ever lead to true epilepsy. It is of considerable importance that we should be able to allay the natural alarm of parents by telling them, after the convulsions have ceased for a sufficient time to allow us to say that they are not epileptic, that there is no chance of their having produced an epilepsy which will develop later.

SYMPTOMS.—Epilepsy may begin in infancy or at any time throughout childhood, but a frequent time for its development is at puberty.

Petit Mal.—The *petit mal* may exist in different degrees of severity. In the mildest form, which may often pass unnoticed unless the attendants are especially on the watch for it, the child stops for a moment in its occupation, whether speaking, eating, or playing, while its eyes become fixed and it assumes a vacant expression. This condition may last for only a few seconds, when the child assumes its former occupation as though it had never been interrupted, and usually is not aware that anything has happened. In other cases this condition lasts a little longer, and slight twitching of the lower part of the face and of the extremities may occur. In other cases, again, the attacks are more severe, the child complains of being dizzy, staggers, has slight convulsive movements, and turns pale, this condition lasting for a minute or more, and being quite marked, but without any total loss of consciousness. Momentary attacks of staggering sometimes occur alone in place of the attacks above described. At times these attacks of petit mal are the only manifestations of the disease, but in severe cases they are apt to be accompanied by occasional attacks of grand mal. They may occur as often as twenty or thirty times a day, or, on the other hand, they may be noticed only once in four or five days, and sometimes they are absent for longer intervals.

Grand Mal.—In the *grand mal* the attacks are of much greater severity. They are sometimes preceded for several hours by a feeling of malaise or general discomfort, but this is not always present. Patients sometimes have notice of the sudden onset of the attack, and such notice immediately preceding the convulsions and forming part of the attack itself is called the *aura*. This aura may be of different kinds. It is most commonly a sense of fulness or oppression in the epigastrium, from which something seems to rise into the throat, and unconsciousness supervenes. It may be, however, a pain or a sensation of numbness, tingling, or other form of paræsthesia in various parts of the body. Sometimes tinnitus is the first symptom. Frequently the patient has no warning whatever of the attack, but falls unconscious with or without a cry. The face becomes congested, and the eyes usually turn upward so that only the whites can be seen. After this follows the stage of tonic convulsions, which is sometimes so short that it is overlooked. Then come the clonic convulsions, which in typical cases are general, although the limbs on one side of the body are sometimes more affected than those on the other side. The movements of the limbs are apt to be very violent, the hands are clinched, the thumbs being flexed on the palms and the fingers closed over them. In many cases the patients froth at the mouth. In the more severe cases the children bite their tongues and pass their urine involuntarily. The duration of such attacks is usually five or ten minutes, but one attack may succeed another with little or no intermission. When the attacks follow one another in this way for several hours the patient is said to be in the *epileptic status*, and his condition as regards life is very serious. After the convulsion ceases the child's breathing becomes stertorous and the limbs are relaxed. Later, and before consciousness fully returns, the child often falls into a deep sleep, and on waking has no recollection of the attack, but complains only of headache and of mental confusion. Attacks often occur in the night, and in this way may be overlooked, the only evidence of them being that the child has wet the bed. In certain cases, when only nocturnal attacks have been present, we often have reason to believe that the disease has existed for considerable periods before its presence was suspected. In some cases in connection with the attacks there is a desire to walk or to run, so that the patient must be closely watched. In this condition children may walk straight against an obstacle, though they are more apt to stop when something comes across their path. Sometimes they walk or run in circles.

The cases of paroxysmal running described by Bullard are at times the early manifestations of an epilepsy which will develop later, although they may also be only the symptoms of hysteria, chorea, and organic cerebral disease.

Epileptic children are liable to bursts of ungovernable anger and violence lasting for hours, in which they may tear and destroy things, bite the mother or nurse, and are apparently for a time under the influence of illusions and hallucinations.

The condition of patients between the attacks is in the lighter cases and in the beginning of the disease usually quite normal. As the disease progresses, however, there is a tendency to mental impairment, and in the more severe cases, in contrast to the lighter ones, we are apt to find some enfeeblement of intellect, which at times may go on to an advanced dementia.

It has been considered by some observers that those cases in which petit mal exists in connection with the more severe attacks are more liable to mental impairment than those in which the grand mal exists alone.

Diagnosis.—As the convulsive attacks occurring in epilepsy cannot be distinguished clinically from similar attacks due to other causes, we are forced to differentiate epilepsy from other diseases by carefully eliminating other causes for the convulsions. We must also wait to see whether the attacks will continue indefinitely, in which case they are more likely to be epileptic.

The diagnosis of epilepsy is made from a continuance of the attacks after a considerable period without evidence of any organic disease or reflex irritation. When the child bites its tongue during the attack and goes to sleep after the convulsion, or when there is temporary mental impairment after the convulsion, we have good reason to state that the convulsions are due to true epilepsy, especially if no symptoms of organic brain disease coexist.

Epileptic are easily distinguished from hysterical convulsions by the presence of consciousness in the latter, at any rate to a considerable extent. Hysterical convulsions in children are not very common, and almost never exist without the presence of other symptoms of hysteria.

Prognosis.—The prognosis of epilepsy for life is, on the whole, favorable, and epileptics may live for many years.

As regards cure, the prognosis in cases beginning in early infancy is very serious. When the disease begins at the age of ten years or later a certain number seem to recover, at least temporarily. Many authorities consider that true epilepsy is never cured, yet undoubted cases exist in which no convulsions take place for years.

Treatment.—The child should be treated at once, in order to avoid continuous shocks to its nerve-centres. Much benefit results from early attention to general hygienic conditions, to diet, and to protection from nervous disturbances.

The management of these cases demands constant watchfulness and tact, so as to regulate the surroundings of the child in such a way as to avoid all source of irritation and nervous excitement. The diet must be regulated according to the especial indications for each patient. A slight gastric irritation apparently produces more serious consequences in epilepsy than irritation of any other part of the body. A vegetable diet is usually indicated, but when the child does not thrive on this it is advisable to give a certain amount of meat. Eggs are usually well borne.

The bromides in some form are, in my experience, the most useful drugs. It is often advisable in giving the bromides to change from one bromide salt to another, a greater effect being thus produced than by the constant use of one of them. Efficacious medical treatment depends for the most part on the graduation of the doses, on the selection of the time for changing them, and on the determination of the intervals for administering them. The best results in using the bromides are obtained by diluting the dose with a large quantity of water, 120 c.c. (4 ounces). As a rule, bromide of potassium has been found to be the most efficient and active of the bromides in cases of epilepsy. In giving the bromides it is well to begin with small doses, 0.12 to 0.24 gramme (2 to 4 grains), three or four times in the twenty-four hours, for the first year, and to double this amount for the second year. The dose should be increased gradually until the physiological action of the drug is noticed.

This treatment, at intervals of one or two weeks, should be carried on for long periods, and from six months to a year after the convulsive attacks seem to have ceased.

The following case represents epilepsy in a girl three years old :

She was healthy at birth, and remained so until she was two months old. At that time she began to have slight convulsive attacks, the cause of which could not be accounted for. During the earlier attacks she appeared to be frightened. She would then scream, and become rigid and unconscious for about fifteen minutes, after which she would sleep three or four hours. These attacks occurred at all hours of the day and of the night. They continued at irregular intervals, but were not so frequent after the first year and a half. During the first year she seemed as bright as any infant of her age, and developed normally.

She was treated with the bromides, and they seem to have been of some benefit, but did not produce a permanent cure.

In the third year her mental condition was much affected. She had never been able to sit alone or to bear her weight on her feet. She could not feed herself, and she understood very little that was said to her. Her head was about the normal size. Her face and eyes had a vacant expression, and she had to be taken care of as though she were an infant in the early months of life.

In this case there was no history of epilepsy or of any especial nervous disorder in the family, nor of traumatism or of any serious disease which could have produced this nervous disturbance. It was a case of chronic epilepsy starting from some unknown cause and resulting in permanent idiocy.

INSANITY.

Insanity in children is very rare. In the ordinary forms of insanity no definite pathological lesion has been found which would account for the symptoms presented. Such changes as have been detected come very late in the disease and seem to be secondary. In paretic dementia, however, we find a special form of cortical interstitial encephalitis.

Instances of mania and melancholia at times occur. Hallucinations, which are a common symptom in the insanity of adults, occur in children usually in connection with the delirium of fever, or more rarely with epilepsy. Insanity is met with in children at any age ; it is extremely rare before puberty, but then becomes more frequent.

The prognosis of insanity in children varies according to its form. Acute mania and melancholia are said to recover generally. True paretic dementia is never known to recover.

IDIOCY.—IMBECILITY.

Idiocy is a condition in which there is a complete and permanent impairment of the mind. Imbecility is a slighter grade of mental deficiency. Both conditions occur in brains which are not fully developed in contradistinction to dementia, in which the faculties are lost in a fully developed brain.

ETIOLOGY.—Idiocy may be congenital, arising from intra-uterine disease, or it may be hereditary in families in whom the parents have had such nervous manifestations as insanity, hysteria, epilepsy, and chorea, or who have been syphilitic or alcoholic. The acquired form may be caused when there is a predisposition to mental disease or to a cerebral lesion produced by traumatic conditions, such as pressure on the head at birth, injury to the head by falls or blows, long-continued convulsions, and intracranial hemorrhages. Idiocy is in some cases also associated with hydrocephalus and microcephalus; it may also be a result of epilepsy, especially when the epilepsy develops early in life and the attacks are frequent.

Acute infectious diseases, especially epidemic cerebro-spinal meningitis, are often responsible for the development of idiocy.

PATHOLOGY.—Idiocy is rarely a primary disease, but usually presents the terminal lesions of many different diseases of the brain. The result of these pathological conditions is usually atrophy, which may be local or general, and may be of intra- or extra-uterine origin. Various congenital deformities, as those of the lips, palate, mouth, hands, and feet, are frequently associated with idiocy.

SYMPTOMS.—The symptoms of idiocy vary according as the individual represents a high or a low grade of this condition. An idiot may have a large head from hydrocephalus, or a small head from cerebral nondevelopment or from cerebral atrophy. Again, idiots may have normally developed crania both as to size and as to shape. In the lower grades there is often some physical malformation in connection with the mental impairment. In the more severe cases of idiocy there is considerable incoördination of the limbs, and the movements of the child are awkward and irregular. In many cases the speech is almost unintelligible. The idiot does not take notice of surrounding objects as does the normal child, and even when the sight and hearing are perfectly normal the impressions made on the senses are deadened. Epileptiform convulsions very commonly accompany idiocy, and play a most important part in the general condition of the patient.

The symptoms which are usually met with, and which enable us to diagnosticate a pronounced case of idiocy, are the vacant expression, the occasional presence of strabismus, the drooping head, the drooling, and the lack of all idea of cleanliness. The teeth are usually decayed. Sometimes the child is so limp that he is unable to bear his weight at all, or will stand held by the hands, with his feet apart, his knees bent, and his trunk leaning forward. The whole body sways to and fro with an oscillating movement and absence of equilibrium. When able to walk alone he walks in a staggering, uncertain way, and falls easily. In many cases, however, the child cannot even sit up alone. The muscles of the neck are often so weak that the head falls over on one shoulder or forward on the chest. The vertebral column fails to support the trunk and bends to a marked degree, and all the muscles are feeble and comparatively useless. Lack of power of attention and lack of memory exist in all cases, and in the higher grades are often the most prominent symptoms.

A special type of idiocy has been described under the term *agenesis corticalis*, in which the size and weight of the brain are normal, but in which abnormalities of development of the fissures of Rolando, of the interparietal fissures, and of the cortical nerve-cells have been recognized. This form of idiocy is associated with blindness and terminates in marasmus.

Diagnosis.—We should be careful in very young children not to confuse slow or retarded mental development with idiocy. There is so much variation in the time at which children walk and talk, that a delayed development of these functions must not be considered to represent a condition of mental impairment. Some children develop so slowly, both bodily and mentally, that they appear very backward in comparison with others of the same age. Children in the first year of their lives may be so seriously affected by some grave disease that their development is prevented from advancing normally, and in comparison with other children of the same age they may be far below the usual grade of intelligence. If, however, we examine this class of cases carefully, we see that, although they are very backward in their development, they are gradually developing, and that they do not represent the condition of complete arrest of development which exists in idiots.

It is well to remember that in rhachitis we are apt to have not only retarded mental development, but a weakness of the extremities simulating paralysis. When both these conditions occur, such cases may sometimes be mistaken for idiots.

Treatment.—The treatment of idiots is essentially comprised under the question of their education. The education of this class of cases should be begun early, usually from the fourth to the sixth year. Much can be done to improve the various defects which exist in each individual. He can usually be taught to co-ordinate his movements, and by attending to his general health his physical condition can often be much improved. In many cases if convulsions are present they can be more or less controlled. Malformations or paralyses can be treated with benefit by apparatus or by operation. The best results in these cases will be attained by placing the children in institutions devoted to the training of idiots. Parents can be told that the association of their children with others who are feeble-minded is not a disadvantage, while it is often a great disadvantage for the children of sound mind in a family to be associated with one who is idiotic. In the large majority of cases, however, they will always have to be supervised during their lives, and, in most instances, after they have advanced to a certain point they are liable to retrograde.

MICROCEPHALUS.

When the head is under a certain size it is called microcephalic. The size which is usually accepted as representing a microcephalic head is from 40.5 to 43 cm. (16 to 17 inches). According to Broca, microcephalus exists when the brain weighs 1049 grammes (35 ounces) in the male, and 907 grammes (30 ounces) in the female. It is generally considered that this microcephalic condition is due to a lack of intra-cranial pressure. Together with the lack of development of the cranial bones there exists in these cases a lack of development or atrophy of the brain, which may be considered either as the cause of the lack of intra-cranial pressure or, as is still believed by some writers, as the result of the external pressure caused by a premature synostosis. Microcephalic children are feeble-minded and usually present the symptoms of a somewhat low grade of idiocy. They not infrequently show signs of weakness of the limbs.

MIRROR WRITING.

An unusual and somewhat striking symptom which at times occurs in severe and, as a rule, chronic cerebral disease is one which is called "mirror writing," which is a condition represented by the individual writing in such a way that the letters can only be deciphered when they are reflected in a mirror. This symptom is usually found when there is cerebral degeneration or among the feeble-minded. The actual pathology of the affection has not yet been determined.

HYSTERIA.

Hysteria is a functional disturbance of the cerebral centres represented, according to Möbius, by a state in which ideas control the body and produce morbid changes in its functions. The name is a misnomer, but it has been adopted so generally that we must use it for the present.

Etiology.—We know very little about the etiology of hysteria. Well-marked instances of the disease occur in early life, usually in the middle and later periods of childhood. An inherited nervous organization or highly exciting surroundings, combined with a lack of proper home discipline, appear to present as likely a field for the disease to develop in as any conditions, such as fright, which apparently, at times, directly lead to it. It is a rare disease in children.

Symptoms.—The mere presence of emotional or imaginative conditions in children does not constitute hysteria. For the existence of the disease it is necessary to have definite symptoms, either a markedly disorganized mental state, paralysis, anæsthesia, or some serious loss of function.

The symptoms are innumerable. Convulsions and paralysis are quite common, while dysphagia, amaurosis, and anæsthesia are met with only in the very severe cases, and are not often seen in America. Anæsthesia is especially interesting as representing a pure type of the disease, and is usually on one side of the body. Children perhaps only two or three years of age affected by hysteria will sometimes allow themselves to be pricked on the anæsthetic side of the face without wincing.

Hysteria in children as usually seen in America is marked by the emotional conditions of the child, and by the presence, in many cases, of a fixed idea relating to its own physical condition. The child believes that it cannot perform certain actions or functions, and hence does not perform them. There probably has often been in the beginning some real difficulty or disturbance of the performance of these functions, such as pain, which has passed away or which is not sufficient to produce the present condition.

The most common symptoms, aside from the mental condition, are *convulsions*, *paralysis*, and *anæsthesia*.

The convulsions are distinguished from those of epilepsy by the absence of loss of consciousness. The patient never seriously injures himself in falling, and does not bite his tongue. He does not sleep after the attack.

The paralysis is often of the spastic form, and may be either hemiplegic or paraplegic. In this form the limbs are rigid and the knee-jerks are exaggerated. It may, however, be of the flaccid variety, with the knee-jerks diminished or absent. It is distinguished from the organic forms of paralysis by the normal reaction of the muscles to electricity, by the absence of atrophy, by the absence of any affection of the sphincters, and at times by the presence of anæsthesia.

When anæsthesia occurs it is usually irregular in distribution, occurring in patches, or else it has the same distribution as in cerebral organic disease. It is often variable, changing more or less from day to day.

Although almost any symptom may occur in hysteria, yet the lack of uniformity in the grouping of the symptoms, and the combination of symptoms which belong to entirely different diseases, are of great aid in making the differential diagnosis from these diseases.

We sometimes meet with an exaggerated hysteria in children. The attacks are represented by screaming, running, jumping, and a feeling of being pulled about; they may last for hours, or for days; their duration, however, is usually long,—at times, with intervals, over a year. No signs of organic disease are found in these cases; they seldom injure themselves, and are finally cured by moral influence, change of scene, and good hygienic surroundings.

Hysteria occasionally causes children to present symptoms of serious disease of the spine and joints. This most often follows some slight injury, but may occur spontaneously.

Prognosis.—The prognosis in cases of hysteria is, as a rule, favorable.

Diagnosis.—Generally, the diagnosis is not so difficult as in adult life, because the child is not able to control its sensations of pain and fear so completely as is possible with adults. In surgical cases, however, in which hysterical affections simulate most closely organic disease of the joints, the diagnosis is often attended by extreme difficulty. The application of strong currents of electricity will usually show that the anæsthesia is not real.

Treatment.—The treatment of hysteria is to break up at once the harmful home surroundings, if such exist, and by means of gentle but firm compulsion to make the child understand that its symptoms are unreal. The various local symptoms connected with the digestion and general health of the child should be carefully treated, as the hysterical symptoms are often largely dependent on conditions of this nature.

The following case represents hysteria in a girl ten years old:

The history of the case was that her parents were living and well, and that there were a number of other healthy children in the family. This child had always been well until eighteen days before she entered the hospital. At that time she complained of headache, and on going to school returned feeling sick and apparently unable to speak. She was said to have been unconscious at times, to have had spasms, and to have been very restless at night. She evidently had had great lack of care in her home life, and had been given only poor food. She showed the evidence of this lack of care in the condition of her skin and her digestion on entering the hospital. A physical examination showed nothing abnormal in connection with the thorax and abdominal organs. The pupils were slightly dilated, but were equal and reacted to light. The knee-jerks were decreased. There was no ankle-clonus. She was apparently unable to walk, and she lay in bed taking no notice of anything, but winked her eyes if anything was thrust towards them. Her hearing did not seem to be especially impaired. She lay in a very limp condition, with the legs drawn up in various positions. Her head kept rolling from side to side, and occasionally was retracted. When asleep her head was retracted so as to make nearly a right angle with the body. It was difficult to feed her, and she would not swallow. Her temperature was 37.2° C. (99° F.), her pulse 66, and her respirations 16. When being examined she cried out a great deal.

She was given plenty of good food, and in three or four days her condition was much improved. She took her food well, but was apparently unable to feed herself. A few days later she showed more intelligence, and on being taken up and dressed it was found that she could sit alone and could walk a little with support. On beginning to walk she threw her legs about wildly, but after being scolded she walked much better. At one time when she was sitting quietly in a chair the visiting physician came into the ward, and she immediately allowed herself to slip from the chair and roll onto the floor, but evidently was careful not to hurt herself. She at this time cried out a great deal, but stopped when no notice was taken of her. She was still unable to speak, and, although she could sit up in a chair, apparently noticed nothing.

Nineteen days after entering the hospital she appeared much brighter, and began to take a slight notice of what was going on about her. When questioned, she moved her lips as if about to speak, but made no sound. She continued to improve slowly, and a few days later said "sister," understood what she was told to do, and attempted to do it. She also walked three steps without being assisted. Some days later it was found that she would repeat almost any word that was said to her, but in a whisper. After this she improved rapidly and began to articulate fairly well, but slowly and with an effort. She also spoke voluntarily two or three times. She could not walk without assistance, as she would put her foot too far forward. She had been very much constipated through the whole attack, but at this time the constipation grew less. A definite training of the arms and legs was then begun by means of passive movements and massage. Under this treatment she greatly improved, and on the thirty-fifth day from the time when she entered the hospital, recovered completely.

HYPNOTIC STATE.

The hypnotic state is an artificial mental condition which can be produced in children as well as in adults. It is supposed to be a temporary abeyance of the powers of the higher cerebral centres. In the ordinary cases the child is thrown into a condition in which the consciousness of his external surroundings is lost. This condition in outward appearance closely resembles sleep, but is produced artificially and can be artificially removed. Thus, the sensation of pain can be temporarily abolished, at least to a considerable extent. For this reason it has been supposed that it might be useful in the treatment of cases requiring minor surgical operations. It has also been advocated by some physicians as a form of treatment in various diseases; but our experience at the Children's Hospital has proved it to be inefficient.

CATALEPSY.

Catalepsy is only a symptom. It denotes a condition, apparently of cerebral origin, in which, together with total or partial loss of consciousness, the limbs assume a peculiar form of rigidity called *waxy*, and remain for a considerable time in any position in which they may be placed. It occurs at all ages, but is very rare in childhood. The youngest case that I know of is that of a little girl three years old, reported by A. Jacobi.

The prognosis and treatment are those of the primary disease. There is no especial treatment for a single attack.

SIMULATED DISEASES.

On the boundary-line between children who evidently are suffering from the need of judicious discipline and those who may be said to have the definite disease hysteria, is a class of cases in which *simulation* appears to play an etiological part. These children are usually in the later period of childhood, and seem to have such perverted functions of their nervous centres as actually to represent pictures of diseases which are easily proved not to be present. Deafness, blindness, pains of all varieties, palpitation, dyspnœa, vomiting, spasmodic attacks of various kinds, and many other symptoms arise, and may persist for long periods.

The best treatment for these cases is at once to show the child that its symptoms are unreal and of no importance.

INSOLATION.

Heat-insolation, or heat-stroke, is a condition apparently represented by a functional disturbance connected with the cerebral circulation and produced by heat. This affection in varying degrees is of somewhat frequent occurrence in children, and is supposed to be accompanied by a hyperæmia of greater or less intensity of the meningeal blood-vessels, with general venous congestion throughout the body. It is met with most commonly in the middle period of childhood, because at that age the child is most likely to be exposed to the influences which produce it.

Symptoms.—The clinical picture of this class of cases is, as a rule, quite characteristic. The child has perhaps been playing on a hot summer's day somewhat more vigorously than usual, possibly romping with an older child of more highly developed nervous resistance, getting intensely excited, and greatly overtaxing its muscular strength. It may be that it has been exposed to the direct rays of the mid-day sun, or it may have been playing in some covered but heated and stifling place. The child's nurse, noticing the extremely flushed condition of its face and head and its excited, sparkling eyes, takes alarm and hurries it to its home. Intense headache soon comes on, and in a few hours delirium may supervene. The skin is hot, dry, and reddened; there may be vomiting in the beginning; the carotids and temporal arteries throb perceptibly. The heart's action is violent, and the temperature is raised to 38.9°–39.4°–40° C. (102°–103°–104° F.); the pulse is much accelerated, perhaps 140 to 150, and is full, but usually rhythmical. The conjunctivæ are congested and the pupils contracted. Photophobia to a greater or less degree is almost invariably present. Beyond this there may be no symptoms except a slight amount of muscular twitching, and in some cases a convulsion may occur if the temperature runs as high as 40° to 40.6° C. (104° to 105° F.). The temperature, however, in accordance with the rule in this disease as in others which occur in children, does not always produce the same or equally severe symptoms. Convulsions may occur as a very common form of nervous explosion when fever and disturbance of the cerebral circulation are present, but, as a rule, this symptom is absent.

Prognosis.—We should be careful as to the prognosis given in these cases. Although they often simulate closely a beginning meningitis, yet they are very amenable to treatment, and should therefore be carefully differentiated from that disease. In very severe cases the children may, of course, die of insolation.

Diagnosis.—The diagnosis from meningitis is based upon the history, the milder grade of the symptoms, except the headache, and finally, in doubtful cases, on the rapid recovery and speedy disappearance of the fever.

Treatment.—The treatment of heat-insolation should be prompt and vigorous. A stimulating enema of salt, one teaspoonful to a quart of water, should first be given. The child should then be placed upon a bed protected by a rubber sheet in a cool, darkened room; a warm mustard pack should be applied to the lower extremities, and the neck and chest gently sponged with water at 25° C. (77° F.) for fifteen minutes out of every hour. Leiter's coil should be applied to the head with water at 5° C. (41° F.); bromide of potassium should be given, 0.3 gramme (5 grains) every hour for four doses; a little iced milk may be taken if the child cares for it, not more than one or two ounces at a time; and complete rest and quiet for at least twenty-four hours are usually indicated. The child should be watched carefully for some days and not allowed to play actively enough to get heated. Great care should be taken for the rest of the summer to protect the child from the direct rays of the sun, as after one attack the cerebral circulation remains in a very sensitive condition for a considerable period.

CONCUSSION.

By concussion we mean clinically a group of symptoms following some physical shock, with its resulting traumatic irritation of the nervous centres. I have met with a number of instances of this nervous phenomenon.

One was the case of a boy, four years old, who fell from a table to the floor. I saw him an hour later, and found that his skin was cool, his pulse about 60. He was nauseated and had been vomiting. No evidence of traumatic injury or proof of an organic lesion could be found. After a few hours the symptoms gradually improved, and he was perfectly well on the following day.

These indefinite symptoms are usually ascribed to the brain as the seat of irritation.

The treatment of a case of this kind is simply by perfect rest and quiet in a darkened room, with hot applications to the feet and abdomen, and small and repeated doses of stimulants given by enemata until the stomach is able to retain them, the treatment being continued until the circulation is normal and the pulse strong.

TEMPORARY AMNESIA.

The following case of temporary amnesia represents a class of nervous disturbance the pathology of which we know nothing of and the causes of which are manifold:

A boy, thirteen years old, while running struck his head against a tree. I saw him three hours later. He had walked home, but was a little nauseated, and was put to bed. I found that he had partial loss of memory and was drowsy, but that he had no especial pain. He was perfectly well on the following day.

TEMPORARY APHASIA.

An instance of suspension of the cerebral function connected with the elaboration of words is illustrated by Demme's case.

A child, six years old, previously well and bright, suddenly lost the power of speech. This phenomenon occurred during an operation for talipes, which was being performed without an anæsthetic. After the operation the child was perfectly well, but was unable to elaborate words until the ninth day, when she began to use the one word "mamma" for everything that she wanted to say. She then gradually increased her vocabulary until the twenty-first day, when her aphasia disappeared entirely, and she developed mentally and physically in a normal manner.

ARRESTED PSYCHICAL DEVELOPMENT.

Arrested psychical development is a term used in speaking of an apparent lack of mental growth which is sometimes met with in infancy. So far as we know, it is a functional and not an organic condition of the brain. Infants with this affection develop both mentally and physically for a variable period, perhaps five or six months, and then continue to develop physically but cease to develop mentally. This condition lasts for a variable period of months, when their mental development begins again, and, although for some time they are backward in comparison with other children of their age, they finally show no trace of an abnormal mental condition.

In some cases the arrest of cerebral development is associated with a condition of physical weakness, so that the power to sit and to walk at the usual age is delayed to a much later period, as the third or fourth year.

Arrested psychical development seems to be rather commonly associated with rhachitis, and may also occur in the course of severe illnesses, but nothing else is definitely known concerning it.

RETARDED SPEECH.

When during the second year the power of speech does not develop with the usual rapidity, it is spoken of as retarded speech.

This lack of power to speak may be from a simple lack of development of certain portions of the brain, or from organic or functional cerebral disturbance. It may also arise from abnormal conditions outside of the brain. The cases which are caused by a lack of development may be of congenital origin, or may be due to an arrested cerebral development produced by a number of causes. These causes are usually connected with some serious interference with the cerebral growth, such as a severe illness. Organic aphasia is like that produced by some organic lesion of the brain such as exists in cases of cerebral paralysis. It may also be connected with the condition of idiocy. The functional aphasia I have already described. It may be produced by many causes, among others the infectious diseases. A child may for a time during a severe illness, and after convalescence has been established, apparently be unable to use the words that it was accustomed to before the illness. I have in a number of cases, however, noticed that the child speaks better than it did before the illness.

Retarded speech may also be caused by such physical defects as disease of the ear resulting in deafness, and from such a physical malformation of the mouth, palate, or vocal cords as to render articulation impossible. In this connection stammering may be spoken of as a cause of retarded speech. When called upon to decide why the child is unable to speak, the previous history should be carefully investigated. In this way organic disease of the brain can be eliminated by means of the absence of the usual symptoms of such disease, especially hemiplegia, and by ascertaining that the child has not had any disease sufficiently severe in its character to interfere with the development of the centres of speech. After determining that the child is not an idiot, the ear and mouth should be examined. If the child is deaf, there is a good reason for his not being able to speak. Even when young children have learned to speak fairly well, if they later become deaf from a disease like scarlet fever, they are very apt to become mute also. When a lesion of the ear has occurred before the child has learned to speak, he almost invariably is found to be a deaf-mute, although there may be no defects in articulation or in his mental condition. It is seldom that any defect in the mouth or throat is found which interferes with articulation, except in cases in which very extensive lesions are present, such as cleft palate, and sometimes enlarged tonsils combined with a high-arched palate and a large adenoid growth. The tongue-tie which the parents usually consider to be the cause of the retarded speech is seldom present. When no symptom of organic, functional, or developmental cerebral disease exists, when there is no physical deformity, and when the child hears well and seems bright and well developed in other ways, the parents, as a rule, may be assured that the speech is merely retarded and will probably develop later.

HEADACHES.

When pain in the head occurs in early life it is to be regarded more seriously than at a later period, as it is more apt to indicate some grave central lesion. The various forms of organic headache which arise in children can be spoken of best as symptomatic of the various diseases in which they occur.

There also appears to be a type of headache which occurs in the later years of childhood irrespective of any definite disease and is often unaccompanied by nausea. These headaches, as a rule, are not of serious import, and are usually classed under the term functional. They occur irregularly, and may be in any part of the head. They are often so severe that the child has to lie down. The intervals between the attacks are variable, and the length of the attacks varies from two to three hours to a day. Of these functional headaches the most frequent form in children is that due to anæmia. It is always, however, wiser to look upon this form of headache as a symptom of some undetermined disease.

Although in many cases headaches are caused by an improper regulation of the diet, yet there is evidently some other cause which we do not recognize in their production, as with exactly the same diet for many months a child will show no symptoms whatever of headache. In like manner, although we know that headaches in children may depend upon constipation, yet this class of cases occurs whetner constipation is present or not. The source of the headache cannot be determined by the part of the head affected.

Migraine also may exist in children, and is characterized by severe pain in the head, sometimes unilateral, sometimes bilateral, accompanied by nausea, dizziness, and generally vomiting. The attacks occur at irregular intervals, and usually last the greater part of a day. They may be brought on by apparently slight causes, such as over-fatigue, eye-strain, or very mild indiscretions of diet, in those predisposed to them. These headaches are markedly hereditary.

Although all these forms of headache are ordinarily very intractable to cure, especially when no bad hygienic surroundings exist which might account for them, and when the child does not lead a sedentary life, yet, as a rule, the attacks have a tendency to lessen and disappear as the child grows older.

One of the most common causes in children is pain caused by strain of the eyes. In all cases of headache in children the cause of which is not evident, a careful examination of the eyes should be made, even though there be no symptoms which point to the eyes themselves.

TREATMENT.—The treatment of headache should be directed to that of the disease which is causing the disturbance when it can be detected, as in anæmia or hyperæmia from various causes, gastric disturbances, the prodromal stages of the acute infectious diseases, malaria, uræmia, and other toxic causes; when, however, no cause can be found, a darkened room and sufficient bromide of soda to allay pain and produce sleep are indicated.

VERTIGO.

Vertigo at times occurs in children. It is a term applied to a condition in which the individual or the objects around him appear to be rolling about. It is called subjective vertigo when the patient himself seems to be turning, and objective vertigo when it is the surrounding objects that appear to move.

Vertigo has a variety of causes. It may be due to organic cerebral diseases, such as tumors of the brain, especially of the cerebellum, and to diseases of the ear and of the eye. It may also be due to circulatory disturbances, as in cardiac disease, and to the stomach, as from improper food, also from tobacco and tea.

The following case illustrates this condition:

A boy, thirteen years old, had had a purulent otitis for several years, but this had healed three years previously, leaving a condition of adhesions and cicatrices with considerable impairment of hearing, but with no trouble of the labyrinth nor any symptoms pointing to it.

He was always strong and well until he was seven years old. At ten years he began to have attacks of dizziness accompanied by seeing white spots. At times he had nausea. A sensation of spinning around or falling subsequently developed, was constant, and increased in severity. He had no other abnormal symptoms except weakness. He slept well, his appetite was fair, and his bowels were regular. He had good hygienic surroundings, did not smoke, and had never lived in a malarial district. He was a close student and led a sedentary life. He had never had any headache. He drank much tea. He was directed to stop drinking tea and to ride on horseback. Within a few weeks after the active exercise had been begun and the tea had been omitted from his diet, the boy ceased to have attacks of vertigo.

PAVOR NOCTURNUS.

The night-terrors of children may occur from a variety of causes, and should not be considered as one disease, but as a symptom of a number of diseases. Any nervous disturbance, whether central or peripheral, may produce so profound an impression on the sensitive cortical cells of the brain that the child's sleep may be disturbed by a cortical irritation.

The following case represents the special form of pavor nocturnus which may be considered *central:*

A boy, six years of age, had always been a delicate, thin, pale child, not caring much for out-of-door exercise, but inclined to remain in the house and to be read to or to have exciting stories told to him. His appetite was poor. He was mentally bright and precocious. Otherwise he appeared to be well, and showed no signs of any organic disease. One evening he was allowed to sit up rather later than usual, and a number of terrifying stories were told to him. He went to sleep as usual, but in about an hour waked up screaming. He was found sitting up in bed looking terrified. His eyes were staring at some invisible object, evidently a picture in his brain and not a reality; he was pointing at this imaginary source of his terror, and kept repeating that it was a black dog. It was impossible to pacify him for about ten minutes, and he did not recognize his mother during the attack. He then became more quiet; the wild look passed from his eyes; he recognized his mother, and soon lay down and went quietly to sleep. The cause of this attack, which is typical of the central form of pavor nocturnus, was evidently an undue excitement of the cells of the cortex in a bright, nervous child before going to sleep. The treatment of a case of this kind is to accustom the child to more exercise in the open air, to prevent his reading anything but the most ordinary and simple books, and to have no stories whatever related to him, especially at night.

The following case represents the *peripheral form* of pavor nocturnus:

A girl, three years old, had been always well and strong; she had a good appetite; was not nervous or excitable; she was fond of playing out of doors, and was not fond of having stories told to her. She was constipated, and had a tendency to overload her stomach. She had eaten a very heavy supper, and on going to bed immediately fell asleep, but soon began to be restless, to throw herself about, to groan, and to grind her teeth. A little later she woke up screaming, and apparently had a certain amount of dyspnœa. She did not recognize her mother, but sat up in bed looking very much frightened and clutching at her throat. Her mother made her drink some warm water, which produced copious vomiting. She then became rational again, recognized her mother, and soon lay down and went to sleep. She had no recollection of the attack on the following day.

The irritation in this case was of the terminal filaments of the pneumogastric nerve in the stomach, causing reflex symptoms of the nervous centres to such an extent that the child was terrified and felt as though she would stifle.

The treatment in cases of this kind should be by moderating the diet and allowing the child to have only a light and digestible supper. The two classes of cases are distinct and their treatment is entirely different. A mixture of both of these forms is frequently met with, between which it is not possible to make a clear distinction.

TREMOR.

Universal or partial tremor is rare in infancy and in early childhood in comparison with later life. It does, however, occur, and is usually significant of an organic cerebral lesion. I have noticed it also in cases of infantile atrophy, in which as recovery gradually took place the tremor disappeared. In this form it appears to be chiefly a symptom of weakness. It may be quite marked as a general symptom, but it is not especially significant, either in respect to the diagnosis or to the prognosis.

TETANY.

With the knowledge which we have at present of this condition we must look upon tetany as a symptom of nervous irritation and not a definite disease. The condition is represented by tonic intermittent paroxysmal muscular contractures varying in duration and intensity. Although the seat of these contractures may be in other parts, such as the neck, face, and thorax, yet the chief and characteristic parts are the hands and arms, legs and feet. The wrist is sharply flexed and turned outward; the thumb in the position of extension is drawn across the palm of the hand beneath the fingers, which are flexed at the metacarpo-phalangeal joints, but otherwise are in the position of extension. The feet are rigidly extended and may be in the position of talipes equinus or of equino-varus. The phalanges of the toes are flexed and extended in the same way as those of the fingers. Tetany as a term should be used only when these characteristic contractures are present, and should not be applied to other conditions of increased reflex excitability. Opisthotonos may occur. The knee- and hip-joints are usually not affected.

ETIOLOGY.—Tetany is a symptom of a number of different conditions, and is not pathognomonic of any one disease. It is at present believed that certain toxins may be developed which affect the central or peripheral nervous system. This is especially liable to occur in gastro-enteric diseases, in rhachitis, in various acute diseases, such as pneumonia, and especially where the hygienic surroundings are bad.

PATHOLOGY.—There is no definite morbid condition with which tetany is associated. The condition is functional rather than organic, and, according to Osler, certain forms depend on the loss of the function of the thyroid gland.

SYMPTOMS.—The peculiar spasmodic conditions described above vary in their duration, and are usually paroxysmal. They may last only a few minutes or for hours or even days. Pain is at times present, especially when the attempt is made to overcome the spasm. The temperature and pulse, according to the exciting disease, are increased. The electrical excitability, mechanical excitability, cutaneous reflexes, and knee-jerks are increased.

Trousseau's symptom (compression of the large nerve-trunks or blood-vessels of the parts affected bringing on the paroxysms) is generally present, and in some cases *Chovstek's symptom* (active contraction of the muscles supplied by the facial nerve produced by a slight tap on the course of the nerve) can be obtained. Laryngo-spasmus occurs in certain cases, especially when rhachitis is present. When the muscles of the trunk are involved, dyspnœa, cyanosis, and even opisthotonos may occur.

DIAGNOSIS.—The peculiar bilateral spasms usually occurring in the course of such diseases as rhachitis or gastro-enteric disturbance, especially when combined with Trousseau's and Chovstek's symptoms, serve to distinguish true tetany from other nervous manifestations of muscular irritability. From tetanus the diagnosis is made by the absence of trismus; from meningitis by the absence of cerebral disturbance; and from cerebral tumor by the bilateral spasm and absence of headache.

PROGNOSIS.—The symptom in itself is not a serious one, the danger depending upon the gravity of the special disease which is causing the tetany.

TREATMENT.—The general nutrition of the child should receive careful attention. Hot baths, chloral, the bromides, and electricity are indicated, and the endeavor should be made to remove the especial cause, which is frequently to be found in the gastro-enteric tract. Thyroid extract should be tried when no primary cause for the tetany can be determined and other treatment is found to be inefficacious.

DENTAL REFLEXES.

The twitching which occurs in children at the time when a tooth is the apparent cause of a certain amount of discomfort and fever should be referred to here as a significant illustration of nervous phenomena from reflex causes. The cases are numerous, but scarcely of sufficient importance to report. In certain instances, however, convulsions of a reflex nature occur at this time and cease when the tooth has assumed its place above the margin of the gum. I have also met with some interesting cases of local œdema arising during the period of dental irritation, and usually spoken of as *angio-neurotic œdema*.

One of these cases was a male infant, fifteen months old, who some months previously, while cutting one of the second molars, had an attack of œdema of the hands, which was not accompanied by irritation or any other symptom, and which passed off after a few hours.

This same boy when the canine teeth were about to come through the gums was again attacked by œdema of the face. This local œdema, as in the previous instance, disappeared quickly.

At times I have seen a local œdema attacking one eyelid, so that the eye could not be closed.

Although we cannot say that irritation from the teeth is necessarily the cause of these conditions, yet they so often arise during the dental period, and not during other periods of childhood, or before the fourth or fifth month, that we can at least say that in individuals of an excessively nervous temperament the irritation which evidently arises in certain cases when the teeth are developing may be sufficient to cause a nervous explosion, which in this sense may be spoken of as of dental origin.

NYSTAGMUS.

By nystagmus is meant a peculiar rhythmical oscillation of the eyeballs, usually from side to side, but in some cases up and down. It may sometimes be dependent on organic disease of the brain, and again may arise from local diseases of the eye, so that a careful examination of the central nervous system and an expert examination of the eyes should always be made. In many cases, however, this condition appears to be purely reflex from various peripheral stimuli, and is not a very uncommon symptom in young children. I have notes of two cases, brothers, who during the dental period showed this oscillation of the eyeballs with no other symptoms. Complete recovery resulted when dentition was complete. The cause, however, of the reflex irritation must always be looked for, and it must not be taken for granted that the chief etiological factor is dentition, for in many cases disturbance of the gastro-enteric tract is present.

GYROSPASM AND SPASMUS NUTANS.

Gyrospasm (rotary movements of the head) and spasmus nutans (nodding) are peculiar movements of the head in young children which are apparently of reflex origin, and are at times associated with nystagmus and strabismus. The chief causes are rhachitis, gastro-enteric irritation, and dentition. The prognosis is usually good except when idiocy is present. The best treatment is with the bromides.

REFLEX SYMPTOMS OF THE EAR.

The reflex connection between the roots of the teeth and the membrana tympani by means of the otic ganglion produces the well-known reflex earache which occurs during the dental period. This has been described on page 634.

REFLEX SYMPTOMS OF THE LARYNX.

In certain cases reflex symptoms occur in the larynx. They are usually noted during infancy rather than in childhood. The affection has been called *laryngospasmus*, and although it is more usual for it to occur in rhachitic children than in others, it is not necessarily confined to rhachitis. It is not in my experience a very common disease, but when met with it is very characteristic.

SYMPTOMS.—The infant is suddenly attacked by a spasmodic contraction of the larynx. This condition may be precipitated by various causes, such as fright and excitement. I have also seen it produced by various peripheral irritations, such as those of the nose. At times the attack is so severe that the infant becomes unconscious and cyanotic. The attack lasts for only a few minutes, and on recovery the infant seems as well as ever. There does not seem to be an inflammatory condition connected with this disease, and apparently it is purely of a reflex nature. In some cases a crowing laryngeal sound will frequently precede and often succeed the more severe stage of the attack.

PROGNOSIS.—The prognosis in cases of laryngospasmus is, as a rule, favorable, although very weak infants may die in an attack.

TREATMENT.—As the infants are usually delicate and of a highly nervous organization, the treatment should be directed to improvement of their general health and to protection from nervous excitement until they have attained an age when their nervous system is less irritable and is in more stable equilibrium. During an attack the treatment is to endeavor to produce relaxation of the spasm by peripheral irritation elsewhere. This is usually done by showering the child on the chest and face with cold water and lightly slapping the back.

Among a number of cases of this kind which have come under my notice was this one:

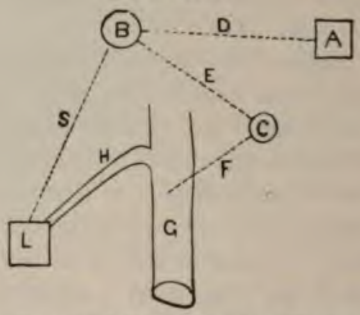
A boy, one year old, had always shown a nervous temperament and had had a number of convulsions when he was cutting his first teeth. With the exception of a light attack of epidemic influenza, he had been well and strong. Following the attack of epidemic influenza, in which the nasal symptoms were prominent, he was left with a very irritable naso-pharynx. He then began to have attacks characterized as follows:

Whenever the nurse, while giving him a bath, attempted to cleanse his nose, no matter how gently, he would immediately gasp, with a catching sound in his breathing, become rigid, draw himself back sometimes almost to the position of opisthotonos, become unconscious and cyanotic, and then after a few seconds the spasm would pass away and he would seem perfectly well again. These attacks continued for some months without apparently harming him, and they then grew less frequent and passed away entirely.

As additional examples of reflex phenomena of the larynx having their origin in the ear may be mentioned the hoarseness which sometimes accompanies the impaction of cerumen in the ears, and which disappears almost immediately after the removal of the mass. Blake reports a case in which a persistent laryngeal cough of several months' duration was immediately relieved by the removal of a bead from the external auditory canal. These cases, as well as those in which there is a reflex cough, can be explained by means of the following diagram, which shows the reflex connection between the ear and the larynx.

The irritation of the sensitive fibres of the auriculo-pneumogastricus distributed in the meatus and in the membrana tympani is reflected along the motor fibres of the superior laryngeal nerve, exciting in the larynx the act of coughing by causing contraction of the crico-thyroid muscle. When the original irritant, either in the meatus or in the membrana tympani, by its continued presence involves the vaso-motor fibres associated with the auricular nerve, they conduct their impression to the ganglion of the pneumogastric, and thence it is deflected through a sympathetic fasciculus proceeding from it to the first cervical ganglion. This again through the nervi molles carries the impression to the external carotid artery, and by its branches to the mucous membrane of the larynx, and as a result of reflected vaso-dilator impressions we may have congestion of the vessels supplying the mucous membrane of the larynx, and perhaps effusion from these vessels.

FIG. 190.



Reflex connection between the ear and the larynx. A, auditory canal, membrana tympani, and middle ear; B, second ganglion of vagus; C, first cervical ganglion of sympathetic; D, auriculo-pneumo-gastric nerve; E, sympathetic fasciculus connecting B and C; F, nervi molles, vasomotor connection with external carotid; G, external carotid; H, laryngeal artery; S, superior laryngeal nerve; L, larynx.

The detailed description of the anatomical conditions underlying these reflex phenomena will be found on page 634.

PAROXYSMAL GASPING.

A condition known as paroxysmal gasping occasionally occurs in young children.

The child, previously quiet, suddenly becomes cyanotic, rolls up its eyes, stops breathing, gasps, and looks as though it were about to die. The seizure is apparently a reflex irritation of the diaphragm, and could be classed under hysteria. These cases respond quite readily to good care, well-regulated food, and relief from the duties of school. In some cases there are no serious symptoms, but mere gasping of light grade.

REFLEX SYMPTOMS OF THE LUNG.

In young infants pulmonary attacks closely simulating the symptoms of asthma occur from gastric irritation of the terminal filaments of the pneumogastric nerve. They are evidently reflex in their character, and are promptly relieved by treatment of the stomach. They are spoken of under the term *asthma dyspepticum*. In cases of this kind it is usually found that the peripheral irritation either arises from the too high percentages of the solid constituents of the milk which is given to the infant, or is caused by the total amount of milk given being too great for the infant's gastric capacity.

SYMPTOMS.—The first symptoms noticed in these cases are the pallor of the infant's face, and a slight cyanosis around the mouth. The respirations then become quickened, and the infant is evidently in great distress. It becomes cyanotic, breathes very rapidly, and often looks as though it were about to die. On examining the chest the lung is found to be resonant, and there is nothing abnormal on auscultation except roughened respiration and a few sonorous râles.

TREATMENT.—An emetic will quickly relieve this condition, which disappears as soon as the stomach is emptied. The attacks are sudden and often recur. After the attack has passed off, the abnormal sounds heard in the lung are found to have disappeared completely, and the infant seems perfectly well again.

Another class of reflex pulmonary symptoms which has at times come under my notice consists of cases in which, from some peripheral irritation elsewhere, marked pulmonary symptoms simulating pneumonia arise.

The first case was a little girl six years old. The first attack occurred at a time when she was having an exacerbation of an attack of subacute purulent otitis media. She had a heightened temperature, 40° C. (104° F.), and rapid respirations (60 in a minute). The alæ nasi were moving slightly, and she showed a certain amount of orthopnœa. Her face was flushed, and she had a short, dry cough. She had a discharge of pus from both ears. On looking at this child one would naturally have said that she had pneumonia. Evidence of this was given by the temperature, the respirations, the alæ nasi, the cough, and the orthopnœa. The lungs, heart, and throat were found on examination to be normal. The pulmonary symptoms were evidently reflex in their nature, as it is believed that in these cases the reflex symptoms are usually produced by the occlusion of the Eustachian tubes.

On inflating the Eustachian tubes with the air-douche her breathing became normal in rate, 24 to 28 in a minute, the alæ nasi ceased to move, the cough disappeared, and the child could lie down with comfort.

REFLEX COUGH.

The nervous connection between the ear and the larynx gives rise at times when there is disease of the former, such as an otitis media, to a persistent cough, which is evidently reflex, and which is relieved only by treatment of the ear.

A little girl, four years old, had an attack of measles which was complicated by an otitis media. She recovered entirely from the measles, and seemed perfectly well, except that the perforation of the membrana tympani had not entirely healed. Somewhat later the cough began. Nothing was found to account for this symptom in the throat, lung, or larynx, except a slightly reddened appearance of the latter from coughing. The cough was intractable to all local treatment until the ear, which had been in the process of healing, again showed signs of increased inflammation. Whenever the ear was discharging, the cough ceased entirely. When the ear was treated and the discharge grew less, the cough began again, and there was an evident reflex connection between the larynx and the ear.

These reflex phenomena continued for some months, the child always coughing when the ear got better and ceasing to cough when the ear got worse. Finally, on the child's being taken to Switzerland and having an entire change of air, its general health was much improved, and the reflex cough passed off. There was no recurrence of this condition in the following ten years.

When there is an irritation in the naso-pharynx a reflex cough often occurs, and is best treated by local applications to the pharynx and naso-pharynx. It is important for the physician to recognize this class of coughs, as he might otherwise be very unsuccessful in treating these cases. Many children are treated with drugs for a cough which is usually ascribed to bronchitis, where no physical signs of irritation can be found in the lung, larynx, or throat, and where the irritation is in the nose or the naso-pharynx. In place of the many drugs usually given in these cases, a simple spray in the nose is indicated.

REFLEX SYMPTOMS OF THE HEART.

Cases of extreme palpitation in children are occasionally met with when nothing organic can be detected, and when no cause, such as tea-drinking, is discoverable. The children are of a highly neurotic temperament, and are usually much influenced by exciting surroundings in their homes.

A boy, ten years old, was subject to fits of great excitement brought on by the most trivial causes, such as preparing to go to school or to take a journey. For some hours before the proper time for starting came he was apt to grow more and more agitated. He would then often be seized with violent palpitation lasting for several hours and forcing him to lie perfectly still on his back. At these times his skin was cool and pale, and his pulse weak and irregular. Nothing abnormal was detected on an examination of the heart or any other organ. The attacks lasted until he was twelve years old, and have never occurred since.

REFLEX SYMPTOMS OF THE STOMACH.

There are a number of reflex conditions connected with the stomach arising from different causes but represented by the same symptom, vomiting. Instances of this condition are those cases of vomiting which arise from irritation of the larynx and pharynx and which are cured by local applications made to these parts.

REFLEX SYMPTOMS OF THE BLADDER.

Reflex spasm of the bladder occurs very frequently in young children, and this condition has been described under enuresis, page 871.

REFLEX SYMPTOMS OF THE VAGINA.

There is almost always a direct cause to be found for the reflex nervous symptoms which arise from vaginal irritation. One of the most common causes is the oxyuris vermicularis, which at times gives rise to extreme and severe symptoms when it has migrated from the rectum. In addition to the local irritation, which causes the child great uneasiness, so that it cannot sit still and is continually moving its legs about, its temperament may be much affected. A child with this trouble is apt to be very fretful, to have violent outbursts of temper, to lose its appetite, and to grow thin.

A little girl, five years old, had the most extreme vaginal irritation. When I saw her she had been affected for several months and was in a very weak condition. At times the irritation seemed to be more than she could bear, so that she would lose all control of herself, would throw herself on the floor, and would have violent spasmodic contractions of the legs. Her sleep was much interfered with, and her whole appearance was that of a child suffering from some serious disease. An examination showed that the oxyuris vermicularis was the cause of the vaginal irritation, and after a few days' treatment directed to the expulsion of the parasite the child ceased to have any irritation and subsequently recovered entirely.

REFLEX SYMPTOMS OF THE RECTUM.

In certain cases reflex symptoms of a most exaggerated type are localized in the rectum. The following cases are illustrative ;

A girl, four years old, had always been remarkably strong and robust, and had never had any especial local trouble with the bladder or the rectum. She was, however, of an excessively nervous temperament, and was surrounded by exciting influences in her home. She began to have spasmodic contractions of the sphincter ani. When she attempted to have a movement of the bowels it frightened her, and she would clutch at any piece of furniture which happened to be near her and try not to have the movement. She would scream and cry out as if she were in much pain. An examination under ether showed nothing abnormal in the rectum or sphincter, such as from pressure or from lesions, and the condition was apparently simply that of spasm. She was treated by the daily dilatation of the sphincter ani with bougies, the size being increased gradually. This was followed by marked improvement, and the rectal spasm passed away.

Although the rectal spasm did not return in this case, yet in its place the child began to have incontinence of urine.

Another child, eight years old, for a whole year was affected by intense irritation in the region of the anus, which prevented her from sitting down for any length of time and kept her in a continual state of irritability. Nothing could be detected during this period which caused these symptoms. No trace of intestinal parasites could be found, and nothing abnormal, either at the anal orifice or in connection with the bowels, was seen, the skin around the anus being in a perfectly normal condition. The child was treated with bromide of potassium for several weeks, and recovered entirely.

CEREBRAL ABSCESS.

Cerebral abscess is a localized purulent encephalitis. It is probably always secondary to suppurative disease elsewhere. It may arise from a suppurative condition of the scalp, but its most common source is some purulent disease of the ear or its surroundings. It is also found as a sequel to traumatism of various kinds resulting in suppuration and in general pyæmia, and it may follow direct traumatic injury to the brain. Cerebral abscess is usually single, except when it is produced by pyæmia.

The abscess may occur in any part of the brain. Cerebellar abscess is not uncommon.

Symptoms.—A cerebral abscess may exist for a considerable time without producing any symptoms which can be recognized during life. In cases in which suppurative disease of the ear exists, a cerebral abscess may be suspected when, in addition to the temperature, which would naturally be raised from this process, the child's general condition becomes worse without any apparent cause, and when indefinite symptoms, such as mental dulness and irritability, arise. The temperature and marked leucocytosis may also suggest the presence of imprisoned pus, and the probability of cerebral disease, in cases where the pus cannot be found elsewhere. Cerebral abscess may, however, exist for a considerable period without rise of temperature, and even with a subnormal temperature. It is apt to be slow in its progress and to cause general constitutional rather than local symptoms. Local symptoms produced by the presence of cerebral abscess are rare. When present, however, they are represented by headache, vertigo, mental dulness, vomiting, and convulsions, and are followed later by coma. When the abscess bursts into the ventricles, symptoms of sudden collapse appear, and death rapidly follows. Tremor and convulsions may occur in cases of cerebral abscess, but neither of them should be considered as in any way symptomatic of this condition.

Prognosis.—The prognosis is very unfavorable unless the disease can be reached surgically.

Treatment.—The treatment should be operative if the abscess can be localized.

CEREBRAL PARALYSIS (Infantile Cerebral Palsies).

In using the term cerebral paralysis it must be understood that it is not intended to describe every disease of intra-cranial origin from which a paralysis may result. We may have a resulting paralysis from many intra-cranial lesions, such as hydrocephalus, cerebral abscess, cerebral tumors, and other causes. These conditions are not included in the class of cases designated as cerebral paralysis or infantile cerebral palsies, which are motor disturbances arising from certain cerebral lesions, occurring in intra-uterine life, during labor or after birth in the first three or four years.

There is much concerning the definite pathology of this class of cases which is still undetermined, so that they will be more clearly understood if discussed as a group and their prominent features compared with each other. This is all the more necessary as the clinical manifestations of all of them are practically the same, and are represented by a spastic paralysis involving one or more extremities in the form of a hemiplegia, a diplegia, or a paraplegia. Monoplegias are rare in comparison with the other forms. Any one of these forms may be accompanied by contractures, choreiform movements, mental impairment, epilepsy, and a number of like nervous symptoms usually spoken of as post-paralytic.

In 225 cases analyzed by Peterson and Sachs, right hemiplegia occurred in 81 cases, left hemiplegia in 75, diplegia in 39, and paraplegia in 30.

ETIOLOGY.—The cause of cerebral paralysis occurring in intra-uterine life has not been definitely determined, and our knowledge concerning this class of cases is very vague. It has, however, been found that instances not infrequently arise in which there is an hereditary history of epilepsy, insanity, or marked neuroses. Traumatism, severe illnesses, and fright in the mother are accepted as other possible causes. According to Sachs, syphilis does not play the important part in the etiology of intra-uterine palsies which has been given to it by some authors.

The cerebral paralysis, which is the result of conditions occurring during labor (birth-palsies), may arise from asphyxia in tedious and prolonged deliveries, which, according to Sachs, are more disastrous than the accidents which at times occur when the forceps are used.

Certain etiological factors are well recognized as causes of the palsies which occur after birth in the early years of life. These are traumatic injury to the skull, fright, the acute infectious diseases, such as measles, scarlet fever, typhoid fever, variola, pneumonia, and cerebro-spinal meningitis. It has also followed acute tonsillitis, severe cases of gastro-enteric disease, pertussis, and, in certain instances, violent convulsions, though in most cases of the latter condition the convulsions are probably caused by, rather than causative of, the lesions producing the paralysis.

In some cases the disease arises in apparently healthy children and without assignable cause.

PATHOLOGY.—In the *intra-uterine* cases the lesions, according to Sachs, are represented by large cerebral defects (*porencephaly*), and also by a condition designated "*agenesis corticalis*," in which there is a defective development of the cellular elements of the cortical and particularly of the pyramidal cells, which is not restricted to any one part of the brain, but involves all parts of the hemispheres about equally. This condition is met with in the family form of idiocy.

In the cases which occur during labor, *birth-palsies*, the primary lesion is usually a meningeal hemorrhage (rarely intra-cerebral), and, as shown by McNutt, more or less diffuse over both hemispheres.

In the cases which occur after birth, *extra-uterine* palsies, as well as in the intra-uterine and birth-palsies, a distinction must be made between the primary lesions and the terminal conditions.

In a series of 78 autopsies in infantile hemiplegia analyzed by Peterson and Sachs, there were found as primary lesions, hemorrhage 23, embolism 7, thrombosis 5, and tubercle 1; while as terminal lesions there were atrophy, sclerosis, and cysts 40, and porencephalus 2. *Porencephalus* denotes a pathological loss of substance, forming cavities or cysts running from the cortex of the brain towards the centre, affecting the motor region and being either unilateral or bilateral. *Sclerosis* consists of a shrinking and hardening of the cerebral tissues, usually more or less strictly localized.

Although the primary lesions of all forms of the infantile cerebral palsies may be caused by embolism or thrombosis, yet these etiological factors are rare in comparison with hemorrhage, which is the most common cause of the primary acute symptoms. This hemorrhage is more apt to be meningeal than cerebral and is usually in the subarachnoid space, thus differing from cerebral hemorrhage in the adult, which occurs more frequently in the region of the internal capsule.

Thrombosis may be a cause where changes have taken place in the cerebral arteries due to hereditary syphilis, and embolism may be the cause in such predisposing affections as valvular cardiac disease.

The pathology, therefore, of the condition as a whole is a lesion of the motor tract followed by atrophy and retarded development of the part affected, and a descending degeneration of the pyramidal tracts and lateral columns of the cord. It is also possible that the primary cause of the disease may be a defective development of the nervous centres.

SYMPTOMS.—If the lesion has been of *intra-uterine origin*, we may get only the later manifestations, just as we do in congenital syphilis. The paralysis in this class of cases is usually diplegic or paraplegic, and mental enfeeblement, amounting often to idiocy, is common. When the paralysis is due to defective cortical development the muscles may be flaccid instead of the characteristic spastic condition of the other forms of cerebral paralysis. If the lesion has occurred at the time of delivery, the primary symptoms are often masked, and the resulting symptoms of the more advanced pathological condition are noticed later.

The diagnostic early symptoms are paralysis, convulsions, rigidity, and stupor; asphyxia and irregular respirations are common symptoms. The symptoms vary with the extent and locality of the lesion. There may be bulging of the fontanelle, changes in the pupils, and oscillation of the eyeballs.

When the disease develops in *extra-uterine* life it is usually acute in its character and is marked by more or less fever, convulsions, and stupor. These early symptoms are merely those of a general nervous explosion following an irritation of the nervous motor centres. They may be the first manifestations of a disease of any kind, or they may occur in the course of one of the diseases of which I have spoken under etiology. If they happen to occur at night and are of short duration, they may be entirely overlooked, and the later symptoms of a cerebral lesion may be the first ones to manifest themselves. The child may die from the severity of these initial lesions before the later symptoms of paralysis have developed by which we can diagnosticate the disease. Screaming, vomiting, and delirium may at times usher in the attack. In the midst of or closely following these primary symptoms come the pronounced indications of a central nervous lesion, represented by hemiplegia (paralysis of an arm and a leg on the same side), paraplegia (paralysis of both legs), or diplegia (paralysis of corresponding parts on the two sides of the body), cases of hemiplegia being the most common. In rare cases we find only one extremity affected (monoplegia). Hemiplegia is by far the most common form.

In addition to the paralysis of the limbs, facial paralysis may occur either in hemiplegia or in diplegia, and, as a rule, spares the upper part of the face, so that the eyes can be closed and the brows raised, thus showing that it is not a peripheral facial paralysis. This form of facial paralysis often disappears early. Strabismus is common.

On examining the paralyzed limb we find a resistance to motion, the deep reflexes are exaggerated, and in most cases there is a feeling of rigidity on the paralyzed side. A few cases of flaccid paralysis have been reported. Sensation, as a rule, is not affected. When the child has come out of its stupor and the convulsions have ceased, it may be found to be aphasic, but aphasia is not so common as in the cerebral lesions of adults. The intelligence is usually impaired, but this depends upon the extent and location of the lesion and the period when it occurred.

The intra-uterine and early infantile cases show the greatest mental disturbance. These children are apt to be very irritable, and, when the lesion is cortical, epileptiform convulsions are quite common. The mind may, however, remain perfectly clear in both the early and late stages of the disease, in spite of a marked hemiplegia. The electrical reaction of the muscles is normal. In the more advanced stages of cerebral paralysis additional symptoms begin to appear. The child learns to walk late, or, if it has already walked, the gait becomes peculiar. Rigidity followed by contracture of the flexor and adductor muscles occurs. In about seventy-five per cent. of the cases of diplegia and paraplegia it comes early and develops oftener than in the adult cases. The rigidity is increased by manipulation or use of the limb, and disappears during sleep. Post-hemiplegic movements follow in a certain number of cases; in others the spastic condition is so pronounced that the patellar tendon reflex and the ankle-clonus cannot be obtained. When walking is attempted, the patient is apt to stand on the toes, the knees knock together, and the spastic rigidity of the muscles produces what is called the spastic gait, represented in its exaggerated form by the cross-legged progression, which is largely caused by the rigidity of the adductors of the thigh. Pes equinus and pes equino-varus are the most common deformities of the foot. When the arm is affected with contractures, the fingers are pressed into the palm of the hand, the hand is flexed, and the arm is flexed at the elbow and held close to the side.

The affected limbs are apt to show some disturbance of their circulation, and some coldness. There are more or less atrophy and shortening of the bone, but to a less degree than in cases of poliomyelitis anterior. In a certain number of cases involuntary incoördinate movements are excited in the paralyzed limbs on voluntary effort (hemiataxia), and are usually designated as post-hemiplegic chorea. There may also be continuous movements (athetosis) of either a partial or a general variety. The sphincters are not affected, whether the case is one of hemiplegia or of paraplegia. Epileptiform convulsions may appear quite early in cases of cerebral paralysis, but may also be delayed for a number of years, so that the possibility of these children becoming epileptic must always be considered.

Diagnosis.—The diagnosis in a marked case of the disease is not difficult, but the determination of the exact lesion is often impossible after the period of onset has passed and we are left with a resulting paralysis. If facial paralysis is present, we can, as a rule, say that the lesion is in the brain; but this rule does not always hold good, as there have been very rare cases in which this paralysis was present when the lesion was in the cord.

The symptoms on which we chiefly rely in making our diagnosis of cerebral paralysis are (1) the distribution of the paralysis, hemiplegic, usually, or paraplegic; (2) rigidity of the muscles; (3) increased tendon reflex; (4) comparatively slight wasting; (5) normal electrical reaction; and (6) mental impairment. Choreic or athetoid unilateral movements associated with a slight increase of tendon reflex point towards a previous cerebral paralysis.

From Poliomyelitis Anterior.—The principal disease from which cerebral paralysis is to be distinguished is poliomyelitis anterior. In contradistinction to the chief diagnostic symptoms of cerebral paralysis just stated we find in poliomyelitis anterior (1) that the distribution of the paralysis is usually monoplegic; (2) that there is an absence of tendon reflex; (3) that there is an absence of rigidity in the early stages; (4) that there is rapid and marked wasting of the affected limbs; (5) that the reaction of degeneration is present; and (6) that there is no mental impairment.

From Idiocy.—In certain cases also a difficulty may arise in correctly understanding the relationship between cerebral paralysis and idiocy. The cerebral lesion is in many cases probably the same, but, according to its extent and location, we may have either (1) a cerebral paralysis alone; (2) a cerebral paralysis accompanied by mental impairment or idiocy; or (3) idiocy without cerebral paralysis. There is a certain class of low-grade idiots in which some impairment of motion exists, apparently due to a mental inability to co-ordinate the muscles of the limbs properly. This may sometimes be accompanied by a diminution of sensation, which seems to be due to a want of perception in the higher nervous centres rather than to any actual lesion of the sensory tract. When the idiot's attention can be kept centred on the limb, the actual sensation does not seem to be much impaired. The differential diagnosis of this condition in idiots from cerebral paralysis is easily made, for it exists only in those cases of idiots in whom the mental development is much impaired, and it is not, as a rule, accompanied by true paralysis, as there is no weakness, but simply incoördination; in these cases also the tendon reflexes are, as a rule, not increased.

From Caries of the Spine.—Cerebral paralysis can be diagnosticated from the paralysis which occurs in connection with caries of the spine, principally by the presence of cerebral symptoms in one case and the prominence of the spinal vertebræ and the rigidity of the spine in the other.

From Syringomyelia.—Rare cases of syringomyelia may be mistaken for cerebral paralysis. The points of differential diagnosis in these cases are that in syringomyelia, although the weakness of the limbs may be so extensive as closely to simulate paralysis, yet the diminution of thermic sensation easily distinguishes it from the normal sensation which is present in cerebral paralysis in cases where the test for sensation can be employed. Syringomyelia, however, is so rare in children that the diagnosis need not be dwelt upon.

PROGNOSIS.—In the intra-uterine forms of cerebral paralysis a large number of infants die at varying periods during the early months of life, so that for some weeks, at least, a prognosis as to life cannot be given. Quite a large number, besides developing epilepsy, also show the condition of idiocy.

A severe lesion may be inferred if convulsions occur in the early weeks and if apathy continues in the intervals between the convulsions. If after a few weeks or months the convulsions are markedly diminished, and if the infant begins to use its legs and to take notice of things, a more favorable prognosis can be given. So long as contractures do not develop a fair use of the extremities may be acquired. Sachs states that in the intra-uterine cases diplegia and paraplegia are more apt to be associated with cerebral deficiency and epilepsy than is hemiplegia.

In the acute extra-uterine cases the prognosis for life in cerebral paralysis is soon determined in the early days of the attack, and depends on the location and extent of the cerebral lesion, but the uncertainty in some cases may last for a number of weeks. On account of the usual meningeal form of the hemorrhage it is less likely to prove fatal in infants than in adults. Entire recovery is rare. In hemiplegia the paralysis will probably improve. The spastic rigidity usually goes on to decided contracture. In some cases no mental change is apparent, in others the mental development is merely retarded, and the child learns to talk some years later than is normal. In a large number of cases, however, the mind is much enfeebled. The occurrence of epilepsy as a result of cerebral paralysis is so common that it should be especially mentioned in this connection, as it makes the prognosis much more serious both as to the degree to which the mental impairment may attain and as to the life of the patient.

According to Gaudard, Osler, Wallenberg, and Sachs, the development of convulsions after an infantile apoplectic attack makes it probable that chronic epilepsy will result, and the prognosis becomes much more grave. Statistics show that epilepsy follows the hemiplegic cases rather more frequently than it does the diplegic and paraplegic, and that it occurs in about half the cases of hemiplegia.

If after a few weeks there is improvement, the prognosis is good; if, on the contrary, there is no improvement for months, it is bad. In some cases, however, after improving for even a year, convulsions may appear and epilepsy develop.

Except in very rare cases, the children can eventually be taught to walk. In many instances, although during infancy there is complete helplessness, later the condition is much improved, and sometimes considerable activity results.

TREATMENT.—In the intra-uterine cases, as it is the later manifestations of the disease which are met with, the treatment should be the same as is indicated in the late treatment of the birth-palsies and of the extra-uterine cases. In acute cases the infant should be kept perfectly quiet, and, if unconscious and unable to nurse, the mother's milk or a carefully modified milk should be given by means of a dropper. If convulsions appear, small doses of the bromides, 0.18 to 0.48 gramme (3 to 8 grains), or chloral, 0.06 to 0.3 gramme (1 to 5 grains), and inhalations of ether are indicated, and in protracted convulsions minute doses of morphia, 0.0006 gramme (0.01 grain).

Although the application of ice to the back of the neck is recommended when a cerebral hemorrhage is suspected, yet in young infants this procedure should be used with great caution, as cold is so apt to reduce their vitality.

The following was a case of cerebral paralysis with left spastic hemiplegia:

The boy was six years old. The delivery was terminated with forceps, but the labor was not a severe one, and he was healthy at birth. He developed normally for two years, and walked when he was eighteen months old. He had convulsions in his third year, and these convulsions occurred again when he was four years old. They were followed by paralysis. He could not use his left hand well, and the grasp of the left hand was less strong than that of the right. The triceps reflex was exaggerated on both sides. The left foot could with difficulty be flexed dorsally. The right knee-jerk was normal, the left increased. He had flat-foot, and walked with his left foot inwardly rotated. He was otherwise well and strong.

The following case represents hemiplegia of traumatic origin, probably hemorrhage:

A girl, four years and nine months old, was brought to the hospital with a history of having fallen from the roof of a three-story building upon a brick sidewalk. She was unconscious. She vomited slightly, and she was found to have an ecchmyosis on the left side of her head. Her pupils were equal and reacted to light. Her respirations were rapid;

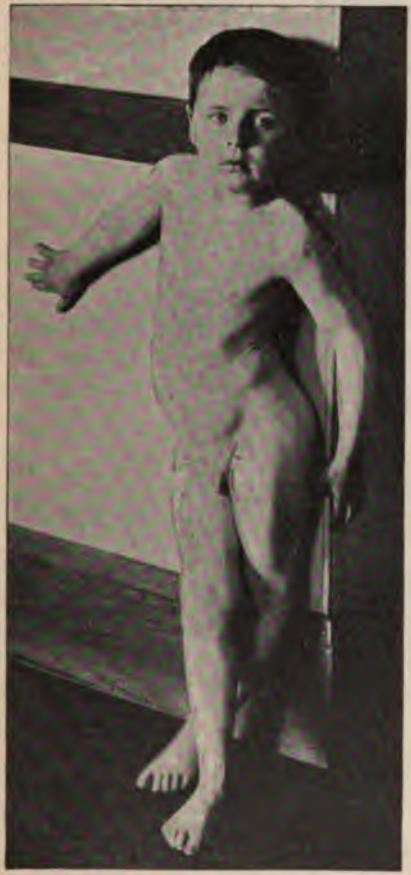
the extremities were cold. She moved all her limbs vigorously. Some clot was found in and about the nostrils. The temperature was 36.3° C. (97.4° pulse was 90, and the respirations were 26. She ground her teeth and cri the night. The muscles of the left arm and leg moved actively.

During the next four days she remained unconscious. Involuntary mi inequality of the pupils, the right pupil not reacting to light, irregular p slight opisthotonos developed as new symptoms.

On the seventh day she appeared brighter, and followed objects with her e pulse was irregular, from 80 to 90.

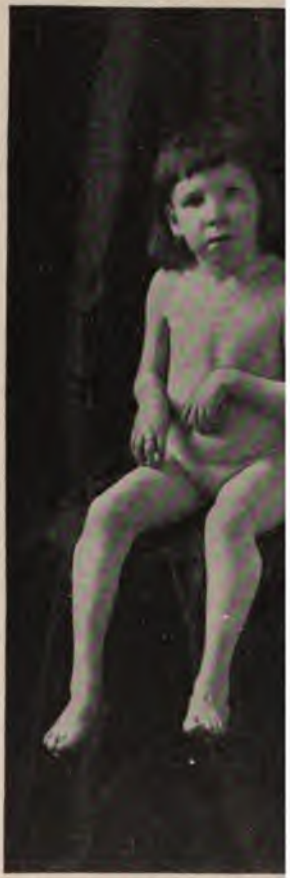
Two days later she made voluntary movements, such as to push objects a her, and gave evidence that she understood what was said to her. From that slowly improved.

FIG. 191.



Cerebral paralysis. Spastic paraplegia. Cross-legged progression. Male, 5½ years old.

FIG. 192.



Cerebral paralysis. Diplegia. extremities affected more than Female, 5 years old.

On the twenty-ninth day from the time when the accident occurred s walk, though with difficulty, as the right leg was very unsteady. One week was discharged from the hospital. She could then use the right arm fairly still walked with some difficulty on account of the weakness of the right l articulation was labored, and her pupils were still unequal.

The following case (Fig. 191) represents a boy, five and a half years old, with spastic paraplegia resulting from a cerebral paralysis and showing cross-legged progression·

The cerebral lesion occurred when he was an infant. Nothing abnormal was noticed about him until he was fifteen months old, when it was observed that he could not walk. He had more or less mental impairment, nystagmus, stiffness of the adductor and flexor muscles, and paralysis of the extensors of the lower extremities. The knee-jerks were much increased, and there was slight ankle-clonus. He walked in the characteristic manner called cross-legged progression.

The following case (Fig. 192) represents a girl, five years old, affected with diplegia resulting from a cerebral paralysis:

She had a good family history. The labor was easy, and was not instrumental. She developed well and was healthy until she was ten months old, when it was noticed that she did not move her arms as she ought to, that she did not use her left arm at all, and that the left leg was not used as well as the right. This condition persisted.

She had strabismus. She could not hold her head up straight. She could not sit up alone or stand. Her head was small and narrow, and had a long antero-posterior diameter. The reflexes were increased. The power of her left arm was much impaired, and there was some contraction of the fingers and elbow of a spastic character. She did not move her left leg well. Sensation was dulled alike in both legs. Her face had an idiotic expression, she was poorly developed mentally, and she could not talk.

She showed the form of spastic cerebral paralysis which is called diplegia, the left extremities being more affected than the right. The face was not involved in this case.

The prognosis of a case like this is unfavorable so far as recovery is concerned, on account of the great mental impairment. Operative treatment is, however, indicated, as at times improvement results in even decidedly idiotic cases.

MYELITIS.

The term myelitis denotes an inflammation of the spinal cord, whether of the gray or of the white matter. Acute myelitis has been used to designate an acute diffuse inflammation of both the gray and the white matter of the cord of non-traumatic origin, and is an affection almost unknown in children.

The term *meningo-myelitis* is used to denote an inflammation of the meninges and of the spinal cord.

Acute myelitis, meningo-myelitis, and hemorrhage into the cord are extremely rare in early life.

INFANTILE SPINAL PARALYSIS (Poliomyelitis Anterior Acuta).

Infantile spinal paralysis is the most frequent and therefore the most important disease which affects the spinal cord, with a resulting paralysis, in infancy and early childhood. It occurs most commonly in the first three years of life. It is rare in the first six months of life. It may occur in later childhood, and very rarely in adults. It is met with more commonly than cerebral paralysis. It is represented clinically by a motor paralysis rapidly leading to atrophy of the muscles affected.

Etiology.—The disease may be primary, in which case it is without any known cause; or it may be apparently secondary to other diseases, such as the acute exanthemata and erysipelas. Traumatism appears occasionally to give rise to the condition. Most of the cases occur during the summer months, and at night rather than during the day. It attacks healthy children as well as unhealthy. It is not hereditary. It may occur in epidemics, and is therefore supposed to be infectious in its origin, but no specific organism has been discovered.

Pathology.—The pathology of poliomyelitis is still not definitely determined. It is at present believed that the gray matter and its ganglion-cells may be involved throughout the entire length of the cord, the process being an interstitial inflammation with secondary changes of a degenerative character in the ganglion-cells. The cervical and lumbar enlargements are most frequently affected.

Goldsheider's investigations show that irritation is present in the walls of the blood-vessels, which leads to their dilatation and to a proliferation of their endothelial elements; the morbid process then extends to the neuroglia and produces a proliferation of its cells. He believes that the changes in the ganglion-cells and in the nerve-fibres are secondary and due to disease of the blood-vessels. Secondary changes also take place in the columns of Clarke, which disappear, and the anterior nerve-roots become smaller than those on the opposite side.

This condition may be confined to the anterior cornua, but in some cases it may involve the spinal meninges. So few post-mortem examinations of the early lesions connected with this disease have been made that we are dependent for our knowledge of it mostly on cases which have been examined a number of months or years after the production of the initial lesion. These later pathological conditions are, however, quite characteristic. The circumference of the limb grows small in comparison with that of the opposite one, being the result of an active muscular wasting and of retarded growth. The bones of the affected limbs are often shorter than those of the other side, sometimes even to the extent of several inches. In certain cases, however, the lengths of the bones seem to be but little affected, although the atrophy of the muscles may be very marked. The anterior cornua of the region affected, which is usually in either the cervical or the lumbar enlargement, are found to be greatly atrophied and many of the large motor cells to have been destroyed. The affected half of the cord may be considerably smaller than the other, and the anterior lateral column may show slight sclerotic changes, chiefly in the pyramidal tract. Accompanying this condition the corresponding anterior nerve-roots are found to be atrophied, and the muscles connected with the region of the cord which is affected atrophy and gradually undergo a fatty and sclerotic change. The fibres are much diminished in size, many have disappeared altogether, and the normal fibres are at times replaced by adipose tissue.

SYMPTOMS.—The onset of the disease in the great majority of cases is acute. Its course is chronic. In the acute form the onset may be preceded for some days by fever and restlessness, but it is apt to appear suddenly with convulsions which, as a rule, are of a milder type than those which occur in cerebral paralysis, are general, and at times absent. The subacute variety of poliomyelitis anterior does not differ from the acute cases in any way in its symptoms, prognosis, diagnosis, and treatment.

Following the acute onset there are at times unconsciousness, lasting sometimes for a number of days, vomiting, general nervous disturbance of the bladder and intestines, and a variety of symptoms of nervous irritability which may represent the prodromata of a number of diseases. The vomiting, when it occurs, accompanies the initial fever, resembles, according to Sachs, the cerebral type, and is not connected with gastric disturbance. Coma may be present, but is rarer than convulsions, and is not usually profound. The temperature is seldom very high, 38.3° to 38.7° C. (101° to 102° F.); it may, however, in certain cases be higher. At times there are no prodromata, but the paralysis is noticed in the morning after a night's rest, although on the evening before the child was seemingly perfectly well. The severity and length of the prodromal symptoms are no indications of the gravity of the lesion or of the prognosis as to recovery. Pain in the paralyzed limb is not an uncommon symptom, but occurs only very early in the disease. The temperature of the limb is lowered, there is vascular sluggishness, and the limb has a bluish, flaccid, undeveloped look. The disease is primarily a motor disturbance, sensation remaining intact. Cerebral symptoms, if present, pass off rapidly with the appearance of the paralysis. The paralysis is usually apt to affect more than one limb in the beginning, but, as a rule, soon becomes monoplegic. The leg is more frequently affected than the arm. Paraplegia in the beginning is not uncommon, and all forms of paralysis may occur. There may also be diplegia, crossed paralysis, and paralysis of the muscles of the back and abdomen. Hemiplegia, so common and almost characteristic of cerebral paralysis, may be present, but is rare in poliomyelitis anterior. The muscles most frequently affected are the extensors, adductors, and supinators. The distribution of the paralysis is usually in groups of muscles. The respiratory muscles may be affected, although rarely. Facial paralysis is so rare that it can almost be said never to occur in uncomplicated poliomyelitis. When the prodromal symptoms have passed off, as they usually do very quickly, the functions of the body are carried on as usual, and the general growth and mental activity are unimpaired. The tendon reflexes disappear in the affected limbs when all the muscles are severely affected. When the paralysis has reached its height, which is usually in a few hours or days, it remains stationary for perhaps from three to six weeks, and then gradual improvement begins, and goes on in certain groups of the paralyzed muscles for six or eight months, leaving other groups paralyzed. These groups again at times recover entirely or remain disorganized, and thus lead later to contractures and deformities. When contractures occur they appear later than do those of cerebral origin.

When paralysis affects wholly or chiefly the gastrocnemii and posterior tibial muscles, the other groups act predominantly, causing dorsal flexion of the foot, so that the child walks on its heel. This condition is termed talipes calcaneus. When, on the other hand, the tibialis anticus and anterior muscles of the leg are most affected, the deformity of talipes equinus occurs; and if the peroneal muscles remain unaffected, there is valgus, while if they are affected with the anterior group, talipes equino-varus occurs. Dislocation of the hip may occur in rare cases of complete paralysis of the leg. Severe cases may show complete flaccidity, and not infrequently the ligaments about the joints are so weakened that the joints become too movable, and the condition called *flail-joint* results. This condition may be present at the hip, knee, or ankle, and sometimes at the shoulder or wrist. Marked atrophy appears in a few weeks. Muscular atrophy, rapid and extreme, is the rule in poliomyelitis anterior, and begins a few weeks after the appearance of the paralysis. Shortening of the bones from arrest of growth may also appear. The surface temperature of the affected limb is lowered, the limb feels cold, relaxed, and lifeless, and the circulation is generally sluggish. Spasmodic movements, except the primary convulsions, are absent.

In rare cases improvement begins immediately after the attack, and goes on to complete recovery (*temporary spinal paralysis*).

In the epidemic form the fever is apt to be high, the distribution of the paralyses extensive, and in a series of ten cases examined by Brackett the sphincters were at times involved, and in the severe cases prolonged hyperæsthesia occurred.

Diagnosis.—In the stage of onset, and until paralysis has appeared, the diagnosis must be held in abeyance. The salient points by which a diagnosis can usually be made are (1) sudden paralysis; (2) loss of tendon reflex; (3) rapid atrophy; (4) cold, flaccid limbs; (5) absence of impairment of sensation; (6) presence of the reaction of degeneration and a diminished reaction to the faradic current.

It is always difficult to diagnosticate poliomyelitis in the initial stage of the disease. At that time pain and sensitiveness of the affected limb may be present, and may lead us to suspect that the disease is rheumatism. The convulsions and unconsciousness which may appear at this stage are so often present in other diseases that they are not of much aid in making the diagnosis of poliomyelitis anterior.

The most reliable test at our command for making the diagnosis of poliomyelitis anterior is the electrical reaction. The normal muscles react to both the faradic and the galvanic current. In applying the galvanic current a quick muscular contraction is noticed both on the opening and on the closing of the negative (cathodal) and of the positive (anodal) pole, but is greater when the cathodal pole is closed. When the galvanic current is applied to the muscles affected by poliomyelitis anterior, the contractions continue, but are slower and less sharp, and the reverse of what takes place in normal muscles occurs. Thus, the anodal closure causes a contraction equal to or greater than that caused by the cathodal closure (*reaction of degeneration*). As the muscles recover there is first a return to the normal galvanic reaction and later to their normal faradic excitability. The diagnosis in young children, however, by means of the galvanic current is practically useless except in the hands of an expert. The faradic excitability begins to diminish within a few days after the onset of the paralysis, and disappears entirely from those muscles which are severely affected.

From Cerebral Paralysis.—Poliomyelitis anterior is most apt to be mistaken for cerebral paralysis, and can be best differentiated from that disease by means of the symptoms which have been described.

TABLE 76.

	Cerebral Paralysis.	Poliomyelitis Anterior.
Motor disturbance	Paralysis. Most common form hemiplegia. Spastic rigidity. Spastic gait. All the muscles of a limb affected.	Paralysis. Most common form monoplegia. Flaccid. Groups of muscles in a limb affected, usually the extensors.
Contractures	Of all the muscles, especially the flexors and adductors.	Of the flexors in the calf.
Spasmodic movements	Athetosis. Post-paralytic chorea. Epileptiform convulsions.	Absent. Convulsions may occur at the onset of the disease.
Sensation	Unaffected.	Unaffected.
Nutrition	Arrest of growth.	Tendency to extreme atrophy coming on early in the paralyzed limb.
Electrical reaction	Normal.	Reaction of degeneration.
Tendon reflex	Exaggerated on the paralyzed side.	Absent.
Speech	Liable to be impaired.	Unimpaired.
Intelligence	Often impaired.	Normal.

From Multiple Neuritis.—The principal points by which multiple neuritis is to be distinguished from poliomyelitis anterior are (*a*) the symmetrical affection of the limbs in the former and tenderness over the nerve-trunks; (*b*) the atrophy in multiple neuritis is not so severe as in cases of poliomyelitis anterior; (*c*) the course of the disease is different, cases of multiple neuritis almost invariably recovering, while severe cases of poliomyelitis do not recover.

From Progressive Central and Progressive Neural Muscular Atrophies.—The diagnosis of these conditions will be found on pages 991 and 992.

From Pseudo-Hypertrophic Muscular Dystrophies.—Pseudo-hypertrophic muscular paralysis in its early stage is not likely to be mistaken for poliomyelitis, but in the later stages of this disease, in which atrophy may occur, it may be necessary to make a differential diagnosis. This will be described on page 994.

From Rhachitis.—In certain cases of rhachitis the power of using the legs is so much affected that the mistake is quite commonly made of supposing that these children are affected by poliomyelitis anterior. The condition in rhachitic children is one of weakness and not of paralysis, and can be distinguished by the normal electrical reaction of the muscles and the lack of pronounced atrophy.

From the Pseudo-Paralysis of Scorbutus.—The pseudo-paralysis which is commonly seen in cases of scorbutus is often mistaken for some organic disease of the central nervous system, with its resulting paralysis. The differential diagnosis from poliomyelitis anterior, however, is not difficult to make, for the involvement of other joints in addition to those of the legs, the presence of pain and tenderness to such a degree that the child cries whenever the limbs are touched, and the normal temperature of the skin clearly distinguish this condition from poliomyelitis, in which disease normal sensation, freedom from pain, and a cold feeling of the limb affected are found.

PROGNOSIS.—So far as a fatal issue is concerned, the prognosis is very favorable. If death occurs, it usually takes place at the end of one or two weeks, and is the result of interference with respiration, which may be caused when the paralysis is extensive. When in the initial stage of the attack cerebral symptoms are prominent and continue for some time, the prognosis is grave. The prognosis is not so good in the epidemic form, which is much more severe in its manifestations.

A second attack of the disease is very rare, and when it occurs it usually comes a few days after the original attack, and manifests itself by an increase of the existing paralysis. The paralysis, as a rule, will not increase when it has been stationary for twenty-four hours. The tendency of poliomyelitis is for a time to improve. Some of the limbs affected recover in the first few days, but if the paralysis persists longer perfect recovery is rare. When there is no improvement after six or eight months the probability is that entire recovery will never take place, although under proper treatment slight improvement may go on for years.

We must remember that, even when untreated, a case of poliomyelitis is very apt to improve for one or two months quite rapidly, then slowly for two or three months, and after a stationary period, contractions, looseness of the joints, and malpositions may begin to be evident and may increase indefinitely.

When proper treatment is carried out, the prognosis is much more favorable, and the period of possible improvement can be extended for some years. According to Bradford and Lovett, there is certainly no leg, however wasted and contracted, that is not amenable to some improvement by operative or mechanical treatment.

Treatment.—The treatment of poliomyelitis by means of drugs has produced such unsatisfactory results that it may be said to be useless. At the onset of the attack the bowels should be freely moved, and convulsions, if present, should be treated symptomatically, as described on page 914.

Although we know of no rational means of treating the primary lesion of poliomyelitis anterior, we know that the results of this lesion, as shown by paralysis of the muscles, are such that the paralysis should be treated at once. The indication is to combat the rapid atrophy which is part of the disease, and to prevent its increase and its later results from proceeding to a degree which would interfere with subsequent repair. To accomplish this, the affected limb should be supported in a normal position and carefully guarded against the stretching of joints, ligaments, and muscles. In addition to this, after an interval of two weeks, gentle massage and the faradic current applied five or ten minutes at a time at least four or five times a week are indicated to keep the affected muscles in the best possible condition and to combat the atrophy which to a greater or less degree occurs. The regular application of heat is also found to be useful where the limb is cold. The muscles are much less likely to contract and deformities to result in properly supported limbs.

The later manifestations of club-foot and other deformities should be dealt with by the orthopædic surgeon. Tenotomy and the transplantation of tendons are of benefit in many severe cases.

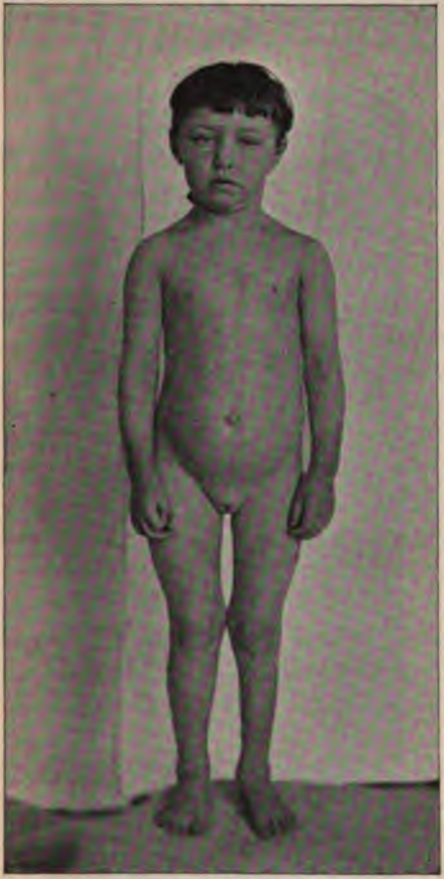
The following case and Fig. 193 represent poliomyelitis in a girl nine years old:

She was perfectly well up to the time of an attack, which came on suddenly and without known cause. She was said to have fallen while she was playing, but no injury of the leg could be detected, although she was carefully examined under ether. The exact date of the attack was not known, but it was not recent. Her general health was reported to have been very good, and she seemed to be bright mentally. She was well developed, and had a good color. Nothing abnormal had been detected on physical examination of the lungs, thorax, abdomen, or other organs. The pulse was regular and of good strength. The left leg showed considerable atrophy, being 4.37 cm. (1¾ inches) smaller than the right in the calf and 2.5 cm. (1 inch) in the thigh. The leg was somewhat cyanotic, and was cold to the touch. There was marked weakness of the muscles below the knee, especially the extensors of the foot and of the toes. When she was lying in bed the movements of the thigh were performed with some strength. On walking she rotated the leg outward, so that the foot was at right angles with the line of motion, and she dragged the toes. The joints were freely movable. Nothing abnormal had been detected in connection with the spine, which presented the condition of a movable lateral curvature, due to the shortening of the affected leg. As the primary lesion probably occurred some years previously, the prognosis was bad as to complete recovery or much improvement.

Fig. 194 represents a girl, two and one-half years old, who had an attack of poliomyelitis anterior of the abdominal muscles.

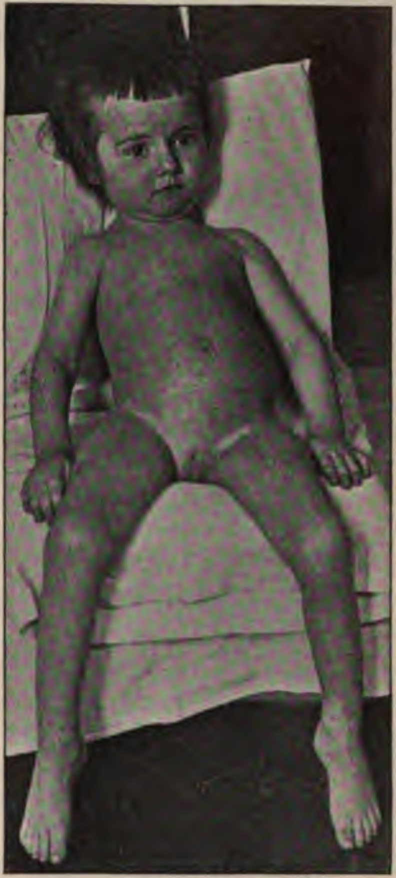
She had a sister who was said to have had an attack of poliomyelitis anterior when she was ten months old. No other history had been obtained about this case, except that she was well and strong until this attack, which occurred six weeks previous to the time when the picture was taken. The onset of the disease was sudden, and was accompanied by high fever, followed in three days by complete paralysis of the muscles of the upper and lower extremities of the body and of the head. The arms and head soon recovered. She was unable to sit up alone, and the abdominal muscles were paralyzed to such an extent on the left side that they were flaccid, bulged out, and did not react normally. The left leg was perfectly flaccid. The knee-jerks were absent. The surface temperature was diminished, and there was atrophy of both legs. Under treatment with electricity and massage, complete recovery took place in this case.

FIG. 193.



Poliomyelitis anterior. Left leg. Female, 9 years old.

FIG. 194.

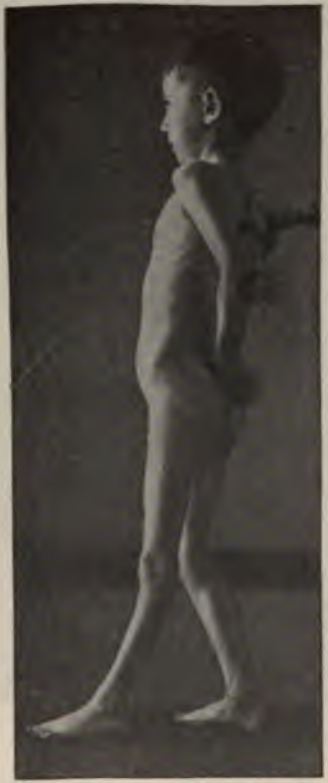


Poliomyelitis anterior. Abdominal muscles, left side. Female, 2½ years old.

Fig. 195 represents the condition of flail leg following an attack of poliomyelitis anterior.

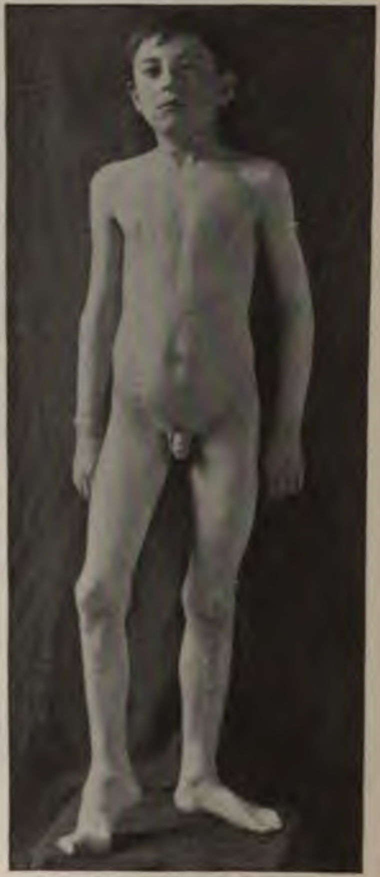
A boy, six and one-half years old, was apparently healthy at birth, and had never had any illness. When he was one year old he was noticed to have some motor disturbance of the left leg. On examination of the leg when he was two and a half years old the surface temperature was found to be diminished, the knee-jerk was absent, and there was an atrophy of 5 cm. (2 inches) of the thigh and 6.5 cm. (2½ inches) of the calf. There was also 3.7 cm. (1½ inches) shortening in the leg. The child walked with a marked limp of the left leg, and there was the condition of flail-joint in his left knee and ankle. This was a typical case of the appearances presented in the advanced stages of a severe case of poliomyelitis anterior.

FIG. 195.



Poliomyelitis anterior. Flail leg, left side. Male, 6½ years old.

FIG. 196.



Poliomyelitis anterior. Talipes equinus on right side. Male, 11½ years old.

The following case represents diplegia caused by poliomyelitis :

The child, a girl of five years, was well and strong until about her third year, when she had an attack of whooping-cough. During this attack she also had some other illness, which was characterized by fever and pain in the back. The loss of power of her legs dated from this time, and is said to have been gradual. She was fairly well developed, and the paralysis had affected both legs and thighs as well as the psoas and iliac muscles. There was marked atrophy, and the reflexes were absent.

The limbs were held apart and were flaccid. If the case had been one of cerebral paralysis there would have been in place of this flaccid condition a contraction of the adductors of the thigh, which would have been apt to hold the limbs closely together.

Fig. 196 represents a boy, eleven and a half years old, with talipes equinus following acute poliomyelitis anterior.

The anterior portion of the foot was flexed at a sharp angle at the medio-tarsal joint. The foot could be easily bent to a right angle, but not beyond. Tense bands of plantar fascia could be felt when the foot was straightened out, but it could be brought into position by the use of considerable force. The length of the legs was equal. There was 1 cm. (about ⅜ inch) atrophy in the right calf and 0.6 cm. (¼ inch) of the right thigh.

This condition of talipes equinus resulted from a contraction of the flexor muscles following an attack of infantile paralysis.

PARALYSIS CAUSED BY CARIES OF THE SPINE.

In cases of paralysis caused by caries of the spine the lesion is essentially a compression of the cord: this is usually slow in its progress, and is not apparently a true inflammation even in the beginning. The condition resulting from compression occurring in the course of caries of the spine may be found in any part of the cord. It is most frequently met with in disease of the dorsal region, though it may occur in the cervical and lumbar regions. In caries of the spine the compression of the cord is not often the result of pressure from the vertebræ, but usually is caused either by an abscess in the vicinity of the diseased vertebræ, or more commonly by a thickening of the meninges.

ETIOLOGY.—When the lesions of the cord are of any considerable extent, ascending and descending secondary degenerations follow after a time. If the process ceases, it leaves a certain amount of sclerosis of the cord at the seat of the disease. This may be very slight, or the cord may be considerably reduced in size and yet may transmit normal nervous impulses.

SYMPTOMS.—The onset of the disease is sometimes quite sudden, but more frequently it is gradual. The symptoms vary according to the part of the cord which is affected.

When the lesion is in the dorsal or the lumbar region the onset is represented by numbness and weakness in the legs. This is quickly followed by a paralysis which may become complete in a short time.

If the lesion is below the level of the sixth dorsal vertebra, the legs alone are affected; if on a level with this point, the abdominal muscles are involved. Sensation up to nearly the level of the lesion may be diminished, or even lost entirely. In regions above the lumbar enlargement the reflex reactions are exaggerated and ankle-clonus soon appears.

When the disease affects the cervical enlargement, or any portion of the cord above, all the extremities are apt to be paralyzed. In severe cases there will be retention of urine, with subsequent incontinence. The bowels are usually constipated, but incontinence of fæces is sometimes present.

In lesions of the lumbar enlargement the knee-jerks will be lost.

Trophic changes in the limbs are not marked, but the muscles are somewhat wasted, and rigidity may develop. Bed-sores are apt to form. The reaction of degeneration is not present.

The characteristic feature of lesions in the dorsal region caused by caries of the spine is a paraplegia.

DIAGNOSIS.—The disease is to be differentiated from poliomyelitis anterior, in which monoplegia is more common than paraplegia, and in which the reflexes are lost and the reaction of degeneration is present. In addition to this means of making a differential diagnosis, the absence of initial fever and prodromata, of disturbances of sensibility, of paralysis of the sphincters, and of a tendency to bed-sores in poliomyelitis anterior is of great aid in differentiating it from the results of caries of the spine, where rigidity of the limbs, increased reflexes, and contractures are prominent symptoms.

The differential diagnosis from cerebral paralysis is more difficult, as the condition of the limbs is similar in both. The diagnosis is made by the absence of all cerebral symptoms, and by the presence of such special symptoms as rigidity and prominence of the vertebræ in caries of the spine.

PROGNOSIS.—The prognosis is in general favorable. A certain number of cases remain uncured, but nearly all recover under treatment, although the condition may persist for months.

TREATMENT.—The treatment of these cases is, as a rule, to be directed to the caries, and consists essentially in perfect rest on a rectangular bed-frame. Massage and electricity are sometimes of assistance when applied to the paralyzed limbs. Forcible correction of the deformity has the effect in many cases of improving the paralysis. When no improvement occurs after several months, laminectomy may be considered.

OBSTETRICAL PARALYSIS.

By obstetrical paralysis, or *paralysis of the new-born*, is meant that form of peripheral paralysis which occurs during the delivery, and which, as a rule, affects the face or one of the extremities. In this sense it is to be separated from injuries to the brain and spinal cord which are produced during the delivery,—in fact, from any paralysis of central origin which may occur in intra-uterine life, either before or at the time of delivery.

ETIOLOGY AND PATHOLOGY.—The cause of this form of peripheral paralysis is most often traction made upon the head of the child during delivery, thus producing a direct injury to the nerves, resulting either in pressure or stretching of the nerves, and in some cases in a rupture of the two upper roots of the brachial plexus. Although this form of paralysis has been known in a number of cases to result from pressure by the forceps during the delivery, yet it has also been met with after an apparently normal delivery, where the pressure did not seem to be especially severe or prolonged.

When the nerves of the face are affected, the resulting lesion is supposed to be from an injury of the facial nerve; and when the arm is affected, the lesion is supposed to be an injury of the brachial plexus or of the nerves in the lower part of the neck. As a rule, the lesion is of one arm.

Symptoms.—A paralysis of this form usually becomes apparent immediately after birth, but may be delayed for several weeks. When the face is affected, it is due, as a rule, to an injury of the seventh nerve, thus producing a peripheral facial paralysis. The peripheral form of facial paralysis is distinguished from the central in that in the former all three branches of the seventh nerve are apt to be affected, while in the latter form only the lower two branches are involved. In the peripheral form, therefore, the eye on the affected side cannot be closed entirely, causing the condition known as *lagophthalmia*, and there is inability to wrinkle the muscles of the forehead on the affected side. In facial paralysis of central origin the muscles of the forehead are not affected, and the ability to close the eye is but little decreased.

Where the paralysis affects an arm it hangs lifeless by the side, with the palm turned backward and the fingers often flexed. The fingers and forearm may be moved, but the movement of the upper arm to any extent is lost.

Diagnosis.—This form of paralysis is to be diagnosticated from cerebral paralysis by the absence of increased reflex irritability and by the distribution of the paralysis. In the cerebral form all the muscles are affected; in the peripheral form, only individual muscles, according to the extent and location of the lesion. It is doubtful whether paralyses of spinal origin occur in the early days of life.

Cases of paralysis of the arm in the new-born should also be diagnosticated from surgical injuries represented by fractures, dislocations, and separation of the epiphyses.

Prognosis.—The prognosis, so far as the face is concerned, is very good, as the paralysis in these instances may last but a short time. We must, however, be somewhat guarded in the opinion which we give concerning these cases, as in some instances the paralysis does not disappear and the muscles of the face are left irreparably injured.

In regard to the paralysis of the arm, in severe cases the prognosis is generally unfavorable, especially if marked improvement does not soon occur, and ordinarily when improvement takes place it is very slow. Most of these cases never recover completely, and even partial recovery should not be expected for a number of years. Shortening of the arm is marked in the later history of the severer cases.

Treatment.—Electricity and massage should be begun after two or three weeks and may have to be continued for a long period. Continuously applied for a number of years they are a very important part of the treatment of these cases, and counteract the atrophy of the muscles from disuse, which must necessarily take place to a greater or less extent.

The following case is illustrative of the effects of obstetrical paralysis:

A boy, two years old, was healthy at birth, but the labor was instrumental. When he was three days old it was found that his left arm was swollen. He was first seen at the hospital when he was seven weeks old. At that time he was able to move his fingers and wrist, but held his arm with the elbow straight to the side and the hand pronated. At the age of two years he was able to make slight movements of flexion of the elbow and slight contractions of the deltoid. Under the use of electricity he showed gradual improvement. He could grasp objects fairly well with his left hand, could flex the elbow completely, and could raise his hand and forearm as far as the nipple. He could raise his right arm with ease to his head, but could not raise the left hand farther than the lower part of the chest.

NEURALGIA.

Neuralgia is a functional affection of the sensory fibres of the peripheral nerves, represented by pain. Neuralgia is rare in infancy and early childhood. It may occur in very different localities, and may be represented by intercostal neuralgia or the various milder forms of flitting pains in different parts of the body.

Sciatica is a form of neuralgia which may occur in children, and is characterized by neuralgic pain and tenderness in the course of the sciatic nerve, especially at its point of exit from the pelvis.

In most cases of neuralgia temporary relief from the pain can be obtained by the use of phenacetine. It can be given in doses of 0.06 to 0.3 gramme (1 to 5 grains), according to the age of the child. Its effects should be guarded by giving brandy and water.

EPIPHYSEAL HYPERÆMIA (Growing Pains).

During the period of middle childhood children at times complain of pains in their limbs, especially the legs. In some cases these pains are probably closely associated with muscular rheumatism, since they may also occur in the joints.

These indefinite pains are usually spoken of as "*growing pains*," but should be distinguished from rheumatism by their slight and evanescent character and by their lasting perhaps only for a few minutes or hours. They are supposed to be induced by fatigue and to be in some way connected with hyperæmia of the epiphyses in the process of their development.

HYDROCEPHALUS.

By hydrocephalus is meant an accumulation of serous fluid in the cranium. This may occur either in the subarachnoid space (*external hydrocephalus*) or in the ventricles (*internal hydrocephalus*).

When the accumulation of fluid represents the only pathological condition present the term primary hydrocephalus is used, while where it follows in the course of other diseases it is termed secondary. Hydrocephalus may also be acute or chronic, congenital or acquired.

Acute Hydrocephalus.—ETIOLOGY.—Acute hydrocephalus may be either external or internal, and is usually a combination of both. It is generally sudden in its onset, and while in some cases it may be idiopathic (*meningitis serosa*), in most cases it is secondary to a number of conditions, such as cardiac disease, pertussis, rhachitis, neoplasms, tumors in the posterior fossa, acute inflammatory processes in the brain and its meninges, and acute febrile diseases, such as typhoid and pneumonia.

SYMPTOMS.—The amount of fluid in these acute cases is, as a rule, not large, 90 to 120 c.c. (3 or 4 ounces), and the symptoms are those which arise in meningitis when there is increased intracranial pressure, such as unequal pupils with sluggish reaction, optic neuritis, rarely convulsions, and cerebral paralyses, which are seldom severe or lasting. In mild cases recovery may take place in a few weeks, but in the more severe forms the increased intracranial pressure may prove fatal.

Both the congenital and the acquired varieties of external hydrocephalus are so rare that they need not be more than mentioned as possibly occurring in certain congenital malformations, in atrophy of the brain (*hydrocephalus in vacuo*), and following cerebral hemorrhage.

The general shape and circumference of the head in infancy and childhood vary normally in the individual to a considerable degree. On the other hand, when these variations in size pass a certain limit or are combined with certain nervous phenomena, they have a distinct pathological significance.

Chronic Internal Hydrocephalus.—The chronic internal variety of hydrocephalus consists of a transudation into the cerebral ventricles. It may be *congenital* (intra-uterine) or *acquired* (extra-uterine). It may follow an operation for spina bifida or be produced mechanically by internal pressure from any cause. It may also represent the termination of an acute inflammatory process.

The chronic form of acquired internal hydrocephalus resembles so closely congenital internal hydrocephalus that we can consider them together, and, so far as the name of the disease is concerned, the term hydrocephalus should be restricted to (1) congenital internal hydrocephalus and (2) chronic acquired internal hydrocephalus. In other words, there exists pathologically a certain class of effusions into the ventricles for which no cause is apparent. When these effusions reach a certain amount the resulting symptoms are quite typical of what is called hydrocephalus, and clinically the term has therefore been confined to cases of this class.

PATHOLOGY.—The anatomical appearance of the brain itself, as a rule, corresponds with and may be accepted as the result of pressure by an intra-ventricular fluid. Fig. 197, page 972, represents the brain of a child who died of congenital internal hydrocephalus, and well exemplifies the pathology of the disease.

The convolutions are flattened and the walls of the ventricles are much thinned by the intra-ventricular pressure, while the ventricles themselves are much dilated. In some parts the cortex is less than 1 cm. ($\frac{3}{8}$ inch) in thickness. The amount of fluid in these cases varies from a few cubic centimetres to three or four litres. The fluid has a specific gravity of about 1004.

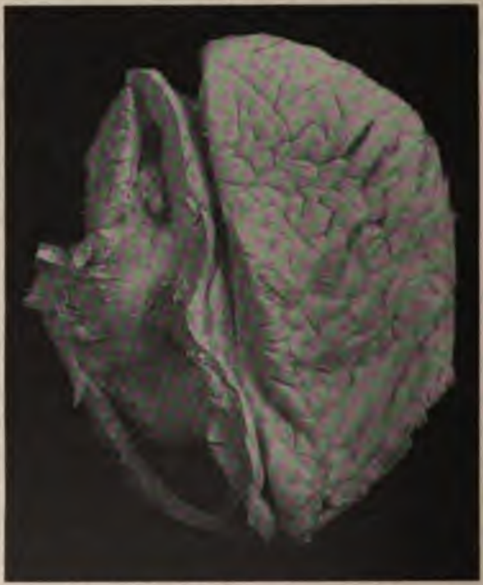
The earlier the hydrocephalic condition begins, the larger will the cranium become. We therefore find the very large heads, as a rule, to be of the congenital variety. The head is at times of such a size as to cause difficulty in the delivery, or the fluid may collect very rapidly after birth, and the head soon assumes the characteristic appearance of hydrocephalus.

Figs. 198 and 199 represent the hydrocephalic skull at three years in comparison with a normal skull of the same age.

The face in these cases of hydrocephalus remains about the same size as it would be normally, but usually looks much smaller from the disproportionate size of the head, which rests upon it like a globe.

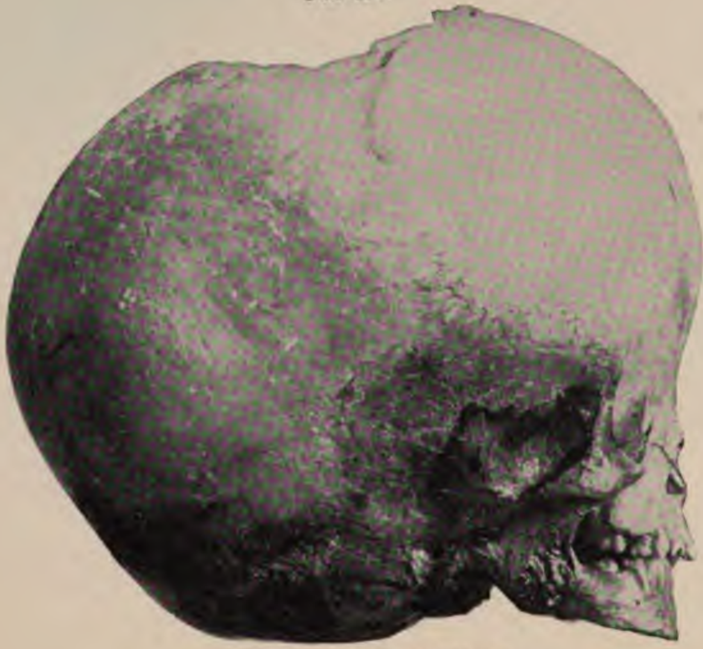
Symptoms.—The symptoms of congenital internal hydrocephalus are essentially those caused by pressure. We naturally, therefore, find the fontanelles bulging and fluctuating, and the bones thin and forced out of position. The temporal and parietal bones diverge as they extend upward, while in the normal skull they ascend almost perpendicularly. This is shown in Figs. 198 and 199. If the disease has existed for some time, the upper wall of the orbit becomes flat and the eyeballs protrude. The intra-cerebral pressure often produces a strong collateral circulation in the scalp and in the forehead, where the vessels appear like tortuous blue cords. Functional disturbances are numerous, and vary in almost

Fig. 197.



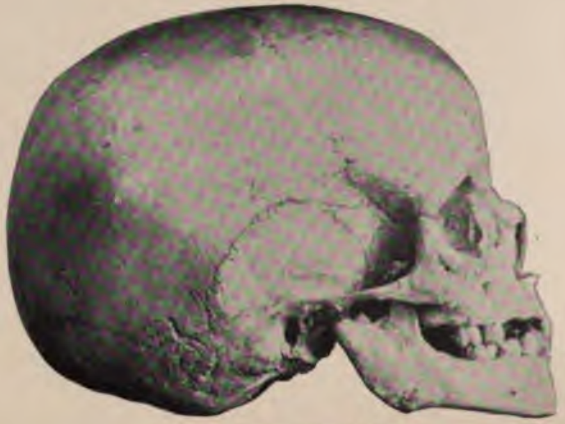
Hydrocephalic brain. Warren Museum. Harvard University.

Fig. 198.



Hydrocephalic skull, child 3 years.

Fig. 199.



Normal skull, child 3 years.

Warren Museum, Harvard University.

every case. As a rule, the children are idiotic, but at times, even in marked hydrocephalus, we find the mental condition normal, even when paralysis is present.

As the various cerebral centres become affected by pressure, symptoms arise corresponding to the parts of the brain which are involved. Among these symptoms are nystagmus and, less frequently, strabismus. The pupils at first are usually moderately dilated. Later they become fixed, and sensibility to light is lost. The hearing lasts for a long time. The ability to walk is interfered with. Partial or general convulsions, paralysis (usually paraplegic), and contractures may occur. Pain in the head is often complained of, but, as a rule, is not so severe as in meningitis. There is difficulty in keeping the head erect, owing to its weight. The digestion is often good, and the appetite usually extreme. The respiration is normal from adaptation. The pulse is usually not retarded. The temperature, as a rule, is normal. The adipose tissue is often abnormally increased.

Diagnosis.—As congenital internal hydrocephalus is almost invariably attended by enlargement of the head and separation of the sutures, the diagnosis is not especially difficult, and is determined by comparing the measurements of the head with those of a normal head of the same age. It must be remembered, however, that hydrocephalus may exist with a small head, and that the general shape and circumference of the head in infancy vary in the individual to a considerable degree. When, however, these variations in size pass a certain limit or are combined with certain nervous phenomena they have a distinct pathological significance. In addition to the enlargement of the head, the symptoms of direct intracephalic pressure make the diagnosis very simple.

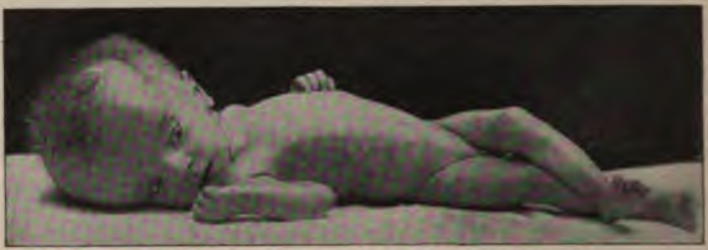
Prognosis.—These congenital cases, as a rule, die before childhood has been reached, but they have been known to live to middle age. Death usually occurs from some intercurrent affection. Complete recovery is very rare.

Treatment.—The treatment of congenital internal hydrocephalus has been varied, but without marked success. When the effusion is not large and is not increasing, moderate pressure with a rubber bandage seems to have a favorable result. When the disease is apparently not in an active state and is characterized by a very slight increase of fluid, aspiration through the anterior fontanelle of a small quantity of fluid at a time has been of temporary benefit. The point of aspiration should be 2 to 3 cm. ($\frac{3}{8}$ inch to $1\frac{1}{4}$ inches) from the median line, so as to avoid puncturing the longitudinal sinus. In this way the condition of the patient is often rendered more satisfactory.

An operation for chronic hydrocephalus presents no technical difficulties. Of course only certain cases are suitable for operation. Moderate effusions should be let alone, also those cases in which a rudimentary development of the brain is suspected. Cases where an operation is especially indicated are both physically and mentally comparatively well developed up to the time when the enlargement of the cranium began. Such children should show the symptoms of direct intra-cranial pressure. They gradually become weak-minded or idiotic. They do not learn to talk, or they quickly forget what they have learned. They may also become totally blind. The power of walking is interfered with. Contractions and partial and general spasms are of ordinary occurrence. Unless the pressure is speedily removed, atrophy of the brain results, and if they live they remain idiots for life. It may, however, be said that no brilliant results as yet have followed the operative treatment of hydrocephalus.

Fig. 200 represents a case of congenital hydrocephalus in an infant seven months old.

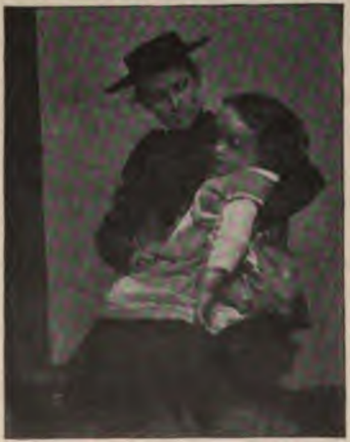
Fig. 200.



Congenital internal hydrocephalus. Male, 7 months old.

It had always been nursed. When it was two days old it had convulsions. Three weeks later it had bronchitis, and accompanying this disease a return of the convulsions, which occurred as often as six or seven times in the day. They were localized in the left arm and left leg. These convulsions lasted for three weeks, gradually growing less severe. There was at this time a certain amount of intestinal disturbance, which, however, disappeared later. There was also a history of a purulent discharge from the ears before the infant was admitted to the hospital. It cried out sharply at night. The measurements of the head were 56.5 cm. (22⅜ inches) in circumference, and 36.7 cm. (14½ inches) from ear to ear over the vertex. The anterior fontanelle was bulging. The eyes were markedly divergent and protruded from their orbits.

Fig. 201.



Congenital internal hydrocephalus. Female, 5 years old.

Fig. 201 represents a child, five years old, with chronic hydrocephalus.

She was nursed by her mother for over a year, and cut her first tooth when she was six months old. She was always well and strong, but high-tempered. She had never had any other disease. When she was five months old she fell from her crib and struck her head, but it did not seem to hurt her especially. Her head was always noticed to be of a peculiar shape. When she was eight months old she fell out of a chair and was stunned, but was not otherwise hurt. She had convulsions from time to time, but her mind had always been bright. She was unable to hold up her head until she was three years old, and had always complained of more or less frontal headache. Her appetite had always been excessive, and her taste for food somewhat peculiar. She gradually grew stronger and was beginning to attempt to walk when last seen. She slept well and her bowels were regular. Her head measured 57 cm. (22½ inches).

The following case represents chronic acquired internal hydrocephalus in a boy four years and eight months old.

The child's parents were healthy; his mother had other healthy children and had had no miscarriages. The child had always been well, measles being the only disease which he had ever had. At the age of six months, while in the process of cutting a tooth, he had three convulsions, from which he recovered entirely. His appetite had always been capricious, but his digestion was good. His bowels had always been regular. He had lately come from a malarial region, where he had lived in a rather damp dwelling for a year.

On May 6 he vomited twelve or thirteen times. The vomiting then stopped, but returned later from time to time. He complained of pain in his stomach, had no fever, and sometimes appeared to feel chilly. His bowels were constipated, and in the beginning of the attack his pulse was slow. He had been subject to night-terrors for some time previous to this sickness. The vomiting had lessened by May 27, and the report of my examination on that date is as follows:

Pulse 60, rhythmical; respirations regular; temperature normal; has had earache lately; no discharge from the ear since he was an infant; the examination of the ear was negative; yesterday morning he had a general clonic convulsion lasting for some time; his tongue is slightly coated; he lies in an apathetic state; though perfectly conscious; he is losing in weight and strength, and has lost his appetite; urine normal. Nothing abnormal is found on examination of the thorax or abdomen. The examination of the eyes, on June 15, showed that there was much swelling of the optic nerve, increased prominence of the retinal vessels, hemorrhages, and neuritis. The child seemed much brighter, and played about. His intelligence was perfectly good; he had had no more convulsions and no paralysis, and seemed perfectly well, except that his pupils were dilated and he was totally blind.

This child came to see me eight years later, when he was twelve years old. He had been and was at that time perfectly well. He was a bright, well-developed, healthy boy. His pupils reacted, but he had never recovered his sight. He weighed 19.8 kilogrammes (90 pounds). His bowels were regular; his appetite was good; his knee-jerks were not increased; his head measured 49 cm. (19½ inches).

THROMBOSIS OF THE CEREBRAL SINUSES.

Etiology.—Thrombosis of the cerebral sinuses is more frequent in infancy and early childhood than in adult life. It is caused by the formation of an ante-mortem clot in one of the sinuses of the brain. As a primary condition it is exceedingly rare. It is usually secondary to some condition which has produced an extreme reduction of the child's vitality, such as profound anæmia, exhausting diarrhœa, or a collection of pus in any part of the body, but especially about the scalp, as in erysipelas. A purulent otorrhœa is perhaps the most common etiological factor. It is not necessary here to do more than refer to the traumatic cases of this disease, in which the ear and the scalp are involved, as in fracture, or in which the thrombosis is caused by compression, as from a cerebral tumor. The pathology of the secondary cases includes the lesions of the different processes which have caused the thrombosis. The thrombosis may take place in any of the cerebral sinuses, and at times may occur in the course of a meningitis. When the thrombus is formed, the venous branches behind the obstruction become distended mechanically, and thus give rise to capillary hemorrhages and softening of the floor of the ventricles. When the thrombosis has taken place in the neighborhood of some inflammatory focus, such as a purulent otitis media, pyæmia may result.

Symptoms.—The symptoms which existed in cases in which this condition has been found on post-mortem examination are not such as to suffice for making a differential diagnosis during life between this and other intra-cranial conditions, such as occur in profound anæmia. When, however, convulsions occur in an atrophic child, especially if there has been chronic trouble in the ear, we can suspect the presence of this condition after carefully differentiating all other causes. Cases of thrombosis of the lateral sinus may be suspected when there are symptoms of a severe purulent affection following a suppurative otitis, with involvement of the mastoid cells, and when there is tenderness over the external jugular vein.

Prognosis.—The prognosis is usually fatal, except when the thrombosis occurs in the lateral sinus and can be relieved by operation. Pitt reports the recovery of a boy ten years old who had chronic otorrhœa, followed by acute symptoms of fever and aural tenderness. Following these symptoms, a week later, he had a rigor, and optic neuritis was developed on the right side. Exploration of the lateral sinus disclosed a clot, which was removed, and the boy recovered.

The following case of cerebral thrombosis came under my observation at the infant hospital:

An infant, nine weeks old, was apparently strong and well up to January 16, when it began to lose in weight. By January 23 it had lost over 200 grammes (6⅔ ounces) without showing any other symptom of disease. By January 27 it had lost 480 grammes (16 ounces). Two days later it was attacked with convulsions and died. The autopsy showed nothing abnormal except a capillary hemorrhage into the ventricles caused by a thrombosis of the straight cerebral sinus.

The pathological diagnosis in this case was that of a sinus-thrombosis of undetermined origin, a condition which is exceedingly rare, and instances of which, established by autopsy, have seldom been reported.

ATHETOSIS.

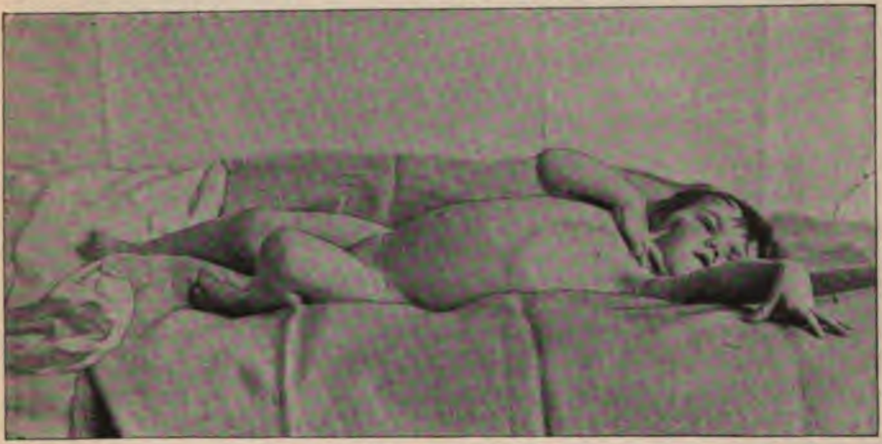
Athetosis is a symptom, and not a disease, and is represented by continuous incoördinate arhythmical movements of the extremities, the face, and the body. This condition may be acquired or congenital. The acquired form may follow cases of hemiplegia or diplegia, in which event it affects the paralyzed limbs. Certain cases of acquired athetosis occur without any accompanying paralysis. In congenital athetosis, and in the acquired form without paralysis, the symptoms usually begin in the first year.

PATHOLOGY.—The pathological condition which exists in cases of athetosis is supposed to be a chronic cerebral irritation in the neighborhood of the basal ganglia and in the internal capsule. The condition as we see it clinically, therefore, is wholly a symptom of some organic lesion of the brain.

DIAGNOSIS.—The diagnosis of *acquired athetosis* is made by the character of the movements. These are continuous, and are distinguished from those of chorea by being vermicular and less spasmodic.

The diagnosis in cases of *congenital athetosis* is not difficult, as in no other disease does an infant present at birth these peculiar movements and this grotesque form of flexion and extension of the fingers and toes. The disease called *congenital chorea*, in which involuntary arhythmical movements exist, is distinguished from athetosis by the character of the movements, which in the former resemble those of ordinary chorea.

FIG. 202.



Congenital athetosis. Female, 2 years old.

PROGNOSIS.—The prognosis of athetosis in regard to recovery is unfavorable. So far as the general health is concerned, the individual may develop fairly well and may live for years.

TREATMENT.—There is no known treatment which has proved to be of benefit in children. As they grow older the training of the affected limbs may be undertaken, but, as a rule, the results are unsatisfactory. Massage and electricity have proved to be of no value.

Fig. 202 represents congenital athetosis in a girl two years old. She never had had any acute disease. She was born after a normal labor, and had received no subsequent injury. She had never talked nor shown much interest in her surroundings, nor had she been able to sit up or hold up her head without support. The bowels had always been regular and the appetite good. She was well developed and well nourished.

The disease was characterized by the continual incoördinate arhythmical movements of the head, trunk, and extremities; these movements were often quite rapid. There were constant flexion and extension of the hands and fingers, the fingers at times being bent backward and assuming most grotesque positions. This phenomenon was also seen in the toes. The expression of the face was not that of ordinary intelligence. The reflexes, on account of the resistance of the child to examination, could not be determined. She was usually irritable, but occasionally smiled slightly, and took some slight notice of those who were near her.

INTRA-CRANIAL TUMORS.

Etiology.—In infancy and early childhood tumors of many varieties may occur in the brain and its meninges. The most common form of intra-cranial tumor is tubercular. The next in frequency are glioma, sarcoma, and glio-sarcoma. The other varieties, such as carcinoma, lipoma, myxoma, and teratoma are very rare; syphilitic gumma, which is so frequent in adults, is exceedingly rare in infancy and early childhood. The parasitic cysts in the brain which occur quite frequently in individuals in other parts of the world, especially in Germany, are seldom met with in this country.

These tumors may be either of intra- or extra-uterine origin. Of these the tubercular is the most common.

Pathology.—The *tubercular* tumors of the brain or its meninges are, as a rule, secondary to a tubercular growth in some other part of the body, or to tubercular disease of some part of the skull, such as the orbit or ear. These tubercular tumors may be single or multiple, the latter being the more common variety. They may be found in any part of the brain or its meninges, and occur with especial frequency in the cerebellum. They may vary in size from a small collection of miliary tubercles to much larger masses. When one or more cheesy masses of a tubercular nature are found in different parts of the brain, the condition is called solitary tubercle. The *gliomata* grow most frequently in the white substance of the brain, but sometimes develop in the gray matter. According to Starr, they grow less rapidly than sarcomata, and never involve the membranes. They are usually primary, but may develop secondary to glioma of the retina (Starr). The *sarcomata* are both of the round-celled and of the spindle-celled variety. Although not quite so frequently found as the gliomata, they are more frequent than the glio-sarcomata or myxomata. They are usually round in shape, develop both in the nervous tissue and in the cerebral membranes, and in both the white and gray matter of the cerebrum and cerebellum. The other varieties of tumor of the brain are so rare that they need not be considered here.

Intra-cranial aneurisms are rare in childhood and are never very large. They are found in the larger arteries of the base of the brain and in the Sylvian arteries.

Symptoms.—The general symptoms vary very much in accordance with the size and vascularity of the tumor, and according as it is growing or has become stationary. In the former case the symptoms are often apt to be more severe than later, when, the tumor having become stationary, the brain-tissue adapts itself to the new conditions produced by the morbid growth. Intra-cranial tumors in infants and in young children are often latent, present no symptoms, and are sometimes discovered only after death. A certain number of cases, on the other hand, present only general symptoms, such as headache, cerebral vomiting, attacks of vertigo, convulsions, and optic neuritis, which cause us to suspect intra-cranial disease, but give an indefinite idea of its location. Again, these tumors may produce local symptoms in addition to the general ones. These local symptoms are represented by paralyses of different kinds, anomalies of sensation, affections of the special senses, and staggering. These later symptoms arise according to the site of the tumor and the parts of the brain which are affected by it, and by means of them we can more or less approximately judge of its situation, size, and rapidity of growth.

Paralyses of the extremities are caused by an affection of the motor cortex, the internal capsule, or any portion of the motor tract on the opposite side of the brain, above the crossing of the pyramids. Staggering or ataxia is suggestive of cerebellar disease, while the involvement of the intra-cranial nerves suggests a tumor of the base of the brain or pressure on these nerves at some point, and more rarely an affection of their nuclei. The tendon reflexes are apt to be exaggerated, but in some cases are normal, and in others are said to be absent. The symptoms of cerebellar ataxia which at times occur when the tumor is situated in the cerebellum consist of a staggering gait resembling that of an intoxicated person, the steps being irregular in length and the body swinging from side to side. The child in these cases has a subjective sense of falling or turning back, and grasps for support or sinks into a chair or to the floor. This form of ataxia is to be distinguished from that which is found in spinal disease, where it is due to an inability to co-ordinate properly the muscles of the lower extremities. This latter form of ataxia is much more regular than the former, each step being insecure and unsteady, but without the violent and sudden reeling, after two or three steady steps, which occurs in the cerebellar form. In young infants a tumor may cause a protuberance of some part of the skull by pushing one of the bones outward.

Diagnosis.—The diagnosis of tumors of the brain must in the great majority of cases be made by elimination. The variety of tumor can be determined most readily by considering the history of the case, as to whether it is tubercular, syphilitic, or otherwise. The diagnosis of a tumor can often be made by the slow and gradual development of the disease. When severe headache and vomiting exist, followed by paralysis, either monoplegic or hemiplegic, especially if this paralysis develops slowly, we should suspect the presence of some form of intra-cranial growth. This suspicion is much strengthened by the presence of optic neuritis or optic atrophy. The presence of localized convulsions in such cases tends to confirm the diagnosis, while if marked ataxia exists we are justified in suspecting cerebellar disease. A normal or only slightly elevated temperature with these symptoms also points to the diagnosis of a cerebral tumor.

PROGNOSIS.—The prognosis of tumors in early life is very unfavorable, no matter what the variety of the tumor may be. Although the patient may for a long time remain wholly unaffected by the morbid growth, he eventually, except in rare cases, succumbs to the disease.

TREATMENT.—Surgical interference in children, as in adults, proves, on the whole, to be the most valuable means at our command for lengthening life in cases of cerebral tumors, but the results have not been what was hoped for when cranial surgery came first into prominence. There is no other treatment which is of any especial benefit in either retarding the growth or curing this class of cases. Where the exceedingly rare form of syphilitic gumma exists, iodide of potassium may be of much value, and is indicated as in adults.

The following case was one of solitary tubercles of the brain:

An infant, thirteen months old, had never had any especial disease, and entered the hospital weak and emaciated. Its mind was clear. Its pulse was weak but regular, and neither slow nor rapid for its age. Its temperature was at times somewhat raised, varying from 37.2° to 38.4° C. (99° to 101° F.). There were no convulsions, and no paralysis or contractures, but merely progressive loss in weight, and finally death.

An examination of the brain showed miliary tubercle of the pia mater at the base of the brain without acute inflammation, which accounted for the lack of acute cerebral symptoms. Of especial interest, however, in the case were some patches of solitary tubercles, 1.2 cm. (½ inch) in diameter, in the left temporal and occipital lobes and in the right frontal lobe of the cerebrum, and also in the lower left cerebellum. There were also caseous tubercles of the post-bronchial lymph-nodes, tubercles of the lungs with a slight amount of broncho-pneumonia, miliary tubercles of the pleura, liver, and spleen, and caseous tubercles of the mesenteric lymph-nodes.

CEREBRAL SYPHILIS.

Intra-cranial syphilis may be either congenital or acquired. According to Bullard, the intra-cranial lesions are essentially the same in both forms.

PATHOLOGY.—Intra-cranial syphilis may be divided pathologically into three forms: (1) diffuse inflammation of the meninges or their neighboring tissues, (2) localized growths or tumors (gummata), and (3) syphilitic endarteritis. In the latter case (endarteritis) there may be local dilatation or local occlusion of the blood-vessels. These conditions are apt to occur simultaneously. When the dilatation reaches an advanced stage a thinning of the arterial walls results, which may lead to rupture of the blood-vessels or to hemorrhage. More common than the hemorrhage, however, is the occlusion of the blood-vessels, which cuts off the blood-supply and acts in the same way as in other cases of thrombosis of the arteries, causing more or less softening and disintegration of the cerebral tissues supplied by them. The arteries of the base of the brain are the ones that are most frequently affected, and there are secondary lesions of the parts of the brain supplied by them.

Symptoms.—The symptoms dependent on these lesions vary in accordance with the pathological condition.

In syphilitic meningitis the principal symptoms are severe headache in various parts of the head, more or less constant, lasting for many days or even weeks, and frequently accompanied after a time by paralysis of some of the intra-cranial nerves, especially of the third or of the seventh. As in other cases of meningitis, the optic nerves may also be affected, and the child shows the general symptoms of a severe intra-cranial affection, such as vomiting, dulness, headache, and localized paralyses.

The localized tumors or gummata present essentially the same symptoms as other forms of tumors of the brain.

The symptoms produced by syphilitic endarteritis vary according to the areas of the brain affected, but the most common ones are the various forms of paralysis of the extremities and sensory disturbances.

Diagnosis.—In regard to the diagnosis of cerebral syphilis in children, the symptoms differ greatly in different cases. The most characteristic group of symptoms, and one which is exceedingly suggestive of intra-cranial syphilis, includes attacks of organic paralysis, central in origin, occurring at intervals of days or months without known cause, and without marked symptoms of either tumor or tuberculosis

The diagnosis of syphilitic meningitis may be made from the occurrence of severe headaches, followed by paralysis of one or more of the motor cranial nerves, and occurring without marked rise of temperature.

Gummata present no especial symptoms from other intra-cranial tumors. The presence of syphilitic lesions elsewhere is our principal ground for making the diagnosis.

Syphilitic endarteritis may be suspected when an acute affection in the neighborhood of the pons or medulla not produced by traumatism occurs in a syphilitic subject, or when acute symptoms suggestive of hemorrhage or embolism occur, and when no other probable cause can be shown, such as cardiac or renal disease.

Prognosis and Treatment.—The results of antisyphilitic treatment in these cases are sometimes striking. The treatment should be pushed to the limit of tolerance.

MENINGITIS.

Although inflammation of the meninges may occur secondarily to disease of the brain itself, and may affect either the dura mater or the pia mater, yet clinically by far the most common condition met with in early life is a *leptomeningitis* forming itself the most important part of the morbid process. *Pachymeningitis* is in early life so rare, except from traumatism or as a lesion of some specific disease such as syphilis, that it need simply be mentioned as of possible occurrence.

The term meningitis may represent a number of diseases, and the symptoms in all are very similar and depend largely on the intensity of the inflammation and on the parts of the brain affected.

The disease may be caused by various specific organisms, such as the tubercle bacillus, the diplococcus intracellularis, the pneumococcus, the typhoid bacillus, the influenza bacillus, and others, or it may be secondary to other diseases or lesions of traumatic origin. These secondary forms are grouped under the head of simple acute meningitis. Tubercular meningitis has been described on page 406 and cerebro-spinal meningitis on page 432.

Simple Acute Meningitis.—ETIOLOGY.—An acute meningitis may arise in the course of a number of infectious diseases, such as erysipelas or ulcerative endocarditis; also after insolation and injuries to the head. It may attack robust as well as debilitated children, and may occur at all ages. It is rare in the first year of life. It is most common in the middle period of childhood. It is especially liable to arise from an extension of the inflammation from disease of the ear, and this in young subjects may take place through the unclosed petrosquamosal suture.

According to Sachs, the cases of idiopathic meningitis which have been reported, which are not due to the specific organisms already described, are probably due to some slight traumatism which has passed unnoticed.

PATHOLOGY.—The pathology of simple acute meningitis is practically, in infants and young children, an inflammation of the pia mater, but there is also usually a mild grade of inflammation of the dura and of the gray matter of the brain. It may be acute or chronic. It is, as a rule, non-purulent in character. The arachnoid may appear somewhat opaque and the cerebral substance œdematous. There is an increase in the cerebro-spinal fluid and the ventricles may be so distended as to correspond to an acute hydrocephalus. The pia of the convexity is most frequently affected, while that of the base may be entirely free, with possibly a slightly increased exudation of lymph in the interpeduncular space.

There is usually an extravasation of white blood-corpuscles near the blood-vessels, which are found on microscopic examination to be slightly engorged. When the meningitis has lasted for a time, the pia, because of the agglutination which has taken place, cannot be removed without injuring the gray matter. In any case of acute meningitis the inflammation is apt to extend downward and to involve the pia mater of the cord.

In the chronic form the pia mater at the base of the brain alone may be inflamed (*basilar meningitis*), or the lesions may be found distributed in small or large areas over the convexity. In these cases the pia mater is thick and opaque, and there is a production of pus, fibrin, and serum, with a formation of new connective tissue. The relative quantity of these inflammatory products varies, and sometimes results in firm and extensive adhesions between the dura and the pia mater. Other conditions which represent the results of chronic inflammation may also be present, but need hardly be referred to here, further than to say that the ventricles of the brain may in this chronic form contain an increased amount of serum and may be dilated. The ependyma also may be thickened and roughened.

Symptoms.—Where acute simple meningitis is secondary to injuries or to other diseases, the characteristic symptoms may, of course, be complicated and even obscured by those of the primary cause. The symptoms, however, are very similar to all the other forms of meningitis which have been described under specific infectious diseases except that they correspond to an inflammatory condition of the convexity rather than to that of the base. The course is often short, sometimes not more than seven or eight days. The disease may, however, prove fatal in forty-eight hours; on the other hand, it may last a number of weeks. When the convexity is chiefly affected, the onset is characterized by intense headache, vertigo, vomiting (usually of a cerebral type), and a high temperature, 40° to 41.1° C. (104° to 106° F.). The respirations are rapid, 30 to 50, and comparatively regular. The pulse in the early stages is rapid, 150 to 170, but is usually regular; later it becomes irregular and slow. Convulsions occur early in the attack. Irritability, listlessness, apathy, and drowsiness subsequently develop. The pupils are contracted at first, but later become dilated; delirium appears; rigidity of the neck is present, and in some cases a marked spastic condition of the lower extremities. The deep reflexes are usually increased; the abdomen is retracted, and the bowels are constipated.

All these symptoms develop rapidly in a few days, and then are intensified as the disease progresses. Blindness (from optic and ocular paralyses) in the form of strabismus and ptosis may appear. A tâche cérébrale is apt to be found, but is not diagnostic. A monoplegic or hemiplegic paralysis having all the symptoms of a cerebral paralysis may develop. In favorable cases the symptoms lessen in severity, and the rigidity, blindness, and paralysis gradually disappear as the child comes out of its coma. In fatal cases the symptoms grow still more severe; the rigidity and retraction of the neck increase; opisthotonos may occur, and the respiration assumes a markedly Cheyne-Stokes type until it ceases at death. Photophobia, contracted pupils, and delirium are present.

Diagnosis.—Great caution should be used in differentiating a simple acute meningitis from the cerebral symptoms accompanying so many acute infectious diseases, for the symptoms are often very similar. Other diseases should therefore first be excluded.

The form of meningitis which is to be especially differentiated from simple meningitis is the tubercular, in which the symptoms correspond more to lesions of the base of the brain as described on page 415.

Prognosis.—The prognosis is very unfavorable. It is possible, however, for the child to recover completely. Perhaps only a changed mental condition will remain, boys appearing effeminate or more easily excited than would be considered normal. Some of the more acute forms affect also the brain, and we find their results in idiocy and contractures.

TREATMENT.—The treatment of simple acute meningitis varies with that of the disease or condition to which it is secondary. The child should be kept in a cool, dark room and protected from noise. In the treatment of meningitis, whether primary or secondary, the indications are to reduce the temperature of the body and to support the general strength until the disease has run its course. The former is accomplished best by sponging the entire body every three or four hours with water at a temperature of from 15.55° to 22.22° C. (60° to 70° F.), and by the application of cold to the head preferably by Leiter's coil. The strength should be supported by the administration of milk, and, when necessary, of stimulants.

The bowels should be freely moved with some purgative such as calomel. Bromide of soda in doses varying from 0.3 gramme (5 grains) to 0.6 gramme (10 grains) should be given according to the age of the child, and every four to six hours according to the severity of the symptoms.

In cases of recovery the greatest care should be taken to keep the child free from excitement for many months, and to insure an absolutely restful life with freedom from physical fatigue. Massage and electricity should be used where there are paralysis and contractions.

ACUTE ENCEPHALITIS.

Although this disease is frequently associated with leptomeningitis, and may occur by simple extension in meningitis due to traumatism or to acute or chronic intoxication, it may also occur, according to Sachs, independently of meningeal lesions, and usually before puberty.

SYMPTOMS.—For a few days headache, dizziness, irritability or depression are noticed. The drowsiness increases to coma, but the loss of consciousness need not be complete, and remissions may be distinct in the first week of the disease. The pupillary reflexes are normal or sluggish; the deep and superficial reflexes are not altered. Rigidity of the neck and opisthotonos, monoplegia, or hemiplegia develop early, and aphasia may or may not be present. Ocular and cranial paralyses occur and simulate basilar meningitis, excepting that the loss of consciousness is not complete. The respiration may become irregular and the pulse slow, rapid, or irregular. The symptoms vary according to the lesions, whether they are of the convexity or base. The disease may last two or three weeks and then prove fatal, or there may be prolonged remissions and complete recovery.

Strümpell, Furbringer, and others have endeavored to establish a form of acute hemorrhagic encephalitis developing commonly after some acute infectious disease, especially influenza, and the pathology is supposed to consist in small and strictly circumscribed inflammatory areas which may be developed in symmetrical parts of the brain.

PROGNOSIS.—The prognosis is bad according to the intensity of the symptoms and the degree of septicæmia present.

TREATMENT.—The treatment should be such as has been described for simple acute meningitis.

BULBAR PARALYSES.

Diseases of the pons and medulla are very rare, but may occur in children with the same symptoms as in adults. They need not be more than mentioned.

Polio-encephalitis, *superior* and *inferior*, and the *acute* and *pseudo-bulbar paralyses* occur so rarely in early life that they hardly have a place in the nervous diseases of childhood.

MULTIPLE NEURITIS.

Neuritis is an inflammation of the peripheral nerves. It is accompanied by pain and tenderness in the affected regions, and in the more severe cases by paralysis and atrophy. Neuritis of a single nerve-trunk or of its branches may be caused by traumatism, cold, or pressure, or may occur in the course of various diseases. In certain constitutional conditions a number of nerves in different parts of the body are affected with neuritis, constituting the disease called *multiple neuritis*.

ETIOLOGY.—Multiple neuritis usually occurs in the course of or subsequent to one of the infectious diseases. Of these diseases diphtheria is the most common, but it is said to follow scarlet fever and measles. A mild form sometimes occurs after typhoid fever. At times multiple neuritis is produced by drugs, such as lead, arsenic, or alcohol. It is not a common disease among children. The epidemic form of the disease, known by the terms kakke and beri-beri, is quite rare in this country.

PATHOLOGY.—The pathological condition in multiple neuritis is an interstitial or parenchymatous inflammation of the nerves. A few nerves may be affected, or the distribution may be general. The nerves of the special senses, however, are rarely affected, and the nerves of the head and face are not usually involved.

SYMPTOMS.—The onset of the disease may be acute or subacute, but in most cases the onset is gradual. It may at the beginning present severe symptoms, such as extreme pain, tenderness over the nerve-trunks, and fever with an accompanying paralysis. On the other hand, the pain in the beginning may be very slight, and the first symptoms noticed may be a gradually increasing weakness of the limbs, while the tenderness may be found only when especially sought for. There may be hyperæsthesia, anæsthesia, numbness, and loss of muscular power. Both the sensory and motor nerves are affected and the condition is symmetrical. After the acute symptoms have passed away the faradic irritability is diminished: the action of the nerves to the galvanic current is diminished, and the reaction of degeneration is present. When the extensors of the leg are affected there is foot-drop, and when those of the forearm are affected there is wrist-drop. All the muscles, although rarely, may be affected and the child may become perfectly helpless. The course of the disease is apt to be a long one of several months, and in the later stages atrophy occurs, while the early hyperæsthesia may give place to a more or less marked anæsthesia, and numbness and various other paræsthesiæ may occur. In mild cases, where only pain and tenderness exist, the knee-jerks are not diminished, and may be even slightly increased, but in the more typical cases of the disease they are absent. Contractures and spasmodic conditions are absent, the paralysis being flaccid. The bladder and rectum are not affected. The temperature is apt to be somewhat raised, and is decidedly so at the onset when the disease is acute. The sensory symptoms are most marked in the beginning of the attack and improve and subside more rapidly than the motor.

DIAGNOSIS.—The diagnosis is made by the same nerve areas being affected by sensory and motor symptoms and by the persistent sensitiveness of these areas. In certain cases poliomyelitis may simulate multiple neuritis, the former being distinguished by the vague pains instead of the pain definitely following the nerve-tracts, by the limited distribution of its paralyses, and by its lack of symmetry. The electrical reactions are much the same in both diseases.

The disease might in some cases be mistaken for a form of acute ascending paralysis, known as *Landry's paralysis*. The latter condition is, however, exceedingly rare in children. Landry's paralysis begins in the legs, involving successively the muscles of the abdomen, thorax, upper extremities, pharynx, larynx, and eyes. The rapid and progressive character of the paralysis and the absence of sensory symptoms are the main points of difference between the two conditions. If Landry's paralysis is made to include cases with sensory symptoms, as is sometimes done, the differential diagnosis from multiple neuritis may at times be impossible.

PROGNOSIS.—The prognosis of multiple neuritis is favorable even when the disease begins with an acute onset accompanied by delirium and high fever, and, although the paralysis may last for many months, the cases usually recover. Exceptionally, however, the paralysis may be permanent, and death may take place from paralysis of the heart or of the muscles of deglutition and respiration. When the reaction of degeneration is present the prognosis for complete recovery is not so good and the case is apt to be prolonged.

TREATMENT.—The treatment is at first by absolute rest in bed, and later with electricity, massage, and strychnine. The primary cause of the disease having been determined, such cause should, if possible, be removed. For instance, if the child lives in a malarial district it should be removed.

The treatment of those cases which follow the acute infectious diseases is symptomatic and hygienic. For the pain the application of heat is the most efficacious remedy. In the acute cases electricity should be begun after three or four weeks; faradism should be used if a reaction is obtained with a moderate current, otherwise galvanism.

Iodide of potassium is indicated in those cases which are caused by lead or arsenic.

In the subacute cases electricity and massage should be employed from the very beginning.

It is safer to wait until the pain and marked tenderness have disappeared before beginning the administration of strychnine.

The following case, a girl, eleven years old, represents multiple neuritis produced by doses of 1 gramme (15 minims) of Fowler's solution given three times a day for some weeks during an attack of chorea.

The first symptoms which were noticed while she was taking the arsenic were that she vomited several times, but this was not supposed to have been caused by the arsenic, and the drug was therefore continued. It was next noticed that the child was unable to walk. Her limbs appeared to be very weak, and there was absence of knee-jerks and ankle-clonus. The sensation of the limbs was normal. A few days later she was found to have tender points over various parts of the legs. The legs then became atrophied. About a month later tender points developed in the arms, and she soon lost the power of using her arms, to such a degree that she had to be fed. At this time, although the arsenic had been omitted for several days, a large quantity of it was found in the urine.

One month later it was found that she could almost support herself without assistance. A little later she walked with crutches, and a month later she could walk without assistance, but with difficulty. The knee-jerks were still absent. She continued to improve, and finally after a number of months recovered entirely.

INSULAR OR DISSEMINATED SCLEROSIS.

Insular or disseminated sclerosis is a chronic degenerative disease of the brain and cord, characterized by multiple distribution of areas of sclerosis.

ETIOLOGY.—The disease appears most frequently as a result of the specific infectious fevers. Heredity is of some importance in the etiology, chiefly inasmuch as it predisposes to a neurotic disposition. The disease is most common in the second and third decades, but many cases in early life, even in infancy, have been reported.

PATHOLOGY.—The sclerotic patches may occur in the brain or cord or in both, and are very irregular in their distribution. The white matter of the brain, the pons, the medulla, and the lateral columns in the dorsal and lumbar regions are most commonly involved. The sclerosed patches consist of proliferated neuroglia, the growth of which destroys the medulla of the nerves with but little destruction of the axis-cylinder.

SYMPTOMS.—The disease is generally slow in its development, and is characterized by tremors on attempting voluntary motions (*intention tremors*); by *scanning speech*, the words being pronounced slowly or with accentuation of the syllables and distinct tremulousness of the voice; by *nystagmus*, a rapid oscillatory movement of the eyes, especially when they are moved laterally; and by *spastic paralysis*, the gait being clumsy and staggering. The muscles are rigid and the deep reflexes increased. Ocular disturbances are common and are similar to those observed in hysteria. Inequality of the pupils and atrophy of the optic nerve may occur. In the majority of cases there is no disturbance of sensation.

As the disease progresses the speech becomes unintelligible, the memory fails, and the muscular weakness and paralysis is more and more marked until the patient is bedridden. Some cases are of more rapid development than others. Some may present bulbar symptoms in which not only the power of speech but of deglutition and respiration is affected.

Diagnosis.—A marked degree of muscular atrophy, paralysis of the bladder and rectum, and changes in electrical reaction are not present in multiple sclerosis, and their absence is of much aid in the differential diagnosis.

Multiple sclerosis may be distinguished from transverse myelitis by the extreme rarity of the latter condition in early life, by its acute onset, the involvement of the bladder, and the sensory symptoms.

The diagnosis from hereditary ataxia is given on page 989. The nystagmus, scanning speech, and intention tremor will serve to distinguish the disease from hysteria, which it may at times resemble. Chorea may be eliminated in the diagnosis by the absence of tremor, by the presence of the characteristic incoördinate movements, and by the absence of nystagmus and true ataxia.

Prognosis.—The disease is incurable, and may last for years. Remission of the symptoms and even improvement may occur, but the general course of the disease is progressive.

Treatment.—Rest in bed, hydrotherapeutics, massage, and general hygiene are desirable in that they alleviate the condition, rendering life more bearable, but no treatment can be said to be in any way curative.

HEREDITARY ATAXIA (Friedreich's Disease).

Hereditary ataxia is a very rare disease, dependent on a slowly progressive sclerosis of the cord at different levels, which involves especially the posterior and lateral columns. It usually occurs in several members of a family, and develops in late childhood.

Symptoms.—The characteristic symptoms are ataxia of the legs and arms, gradual loss of muscular power, with atrophy of the muscles, loss of the knee-jerks, disturbance of speech, and, in the late stages, nystagmus, muscular contractures, from muscular paralyses, and complete helplessness, with mental impairment.

A *cerebellar form* of the disease has been described, characterized by an increased knee-jerk, loss of ocular accommodation, light reflexes, and absence of deformities.

DIAGNOSIS.—The disease is differentiated from tabes dorsalis by the absence of the crises and Argyll-Robertson pupil of tabes, by the hereditary character of the affection, and the extreme rarity of tabes in early life.

It is differentiated from multiple sclerosis by the absence of increased reflexes, intention tremors, spastic gait, and ocular palsies.

PROGNOSIS AND TREATMENT.—The prognosis and treatment of hereditary ataxia is always unfavorable, and no treatment is of benefit except for the relief of symptoms.

LOCOMOTOR ATAXIA.

Locomotor ataxia, or tabes dorsalis, is almost unknown in childhood. The very rare cases resembling it probably represent entirely different lesions from those which are found in cases of the disease occurring in adults.

SYRINGOMYELIA.

Syringomyelia is exceedingly rare in early life. It is regarded as a gliosis, a development of embryonal neuroglia tissue about the central canal of the spinal cord, in which hemorrhage and degeneration takes place with the formation of cavities.

In this disease we usually find a diminution of sensation to heat and cold, according to the site of the lesion, which is commonly a point in the upper dorsal or the lower cervical region. There is apt to be a weakness of one or both arms, accompanied by marked wasting. There is also usually some weakness in the legs. The reflexes are increased, and a spastic condition is likely to result. These symptoms are usually accompanied by marked lateral scoliosis.

Syringomyelia is an incurable disease, and the treatment is therefore usually limited to correcting, if possible, the lateral curvature which frequently accompanies it.

HEREDITARY SPASTIC PARALYSIS.

The term hereditary spastic paralysis has been applied by Sachs to an hereditary disease characterized by spastic rigidity, chiefly of the lower extremities. He recognizes two types of the disease. One is of *spinal type*, a spastic paraplegia with contractures and increased reflexes, due to interference with the pyramidal tract in the lateral column of the cord. Another type, the *cerebral type*, develops at about four or five months of age. It is characterized by increasing mental dulness, defective vision, ending in blindness, nystagmus, and gradual physical deterioration. Convulsions are never present. The symptoms may continue for one or two years, but end fatally. The treatment is symptomatic.

PROGRESSIVE CENTRAL MUSCULAR ATROPHY.

Progressive muscular atrophy of central origin, also described as the *Aran-Duchenne* type, develops in the great majority of cases in adult life after the twenty-fifth year. A few cases, however, have been reported in young children, notably two by Hoffman, one of which occurred in a girl four years of age, and another in her brother, in whom the symptoms began at about the same age.

Various forms are recognized in adults and are known as *amyotrophies*, by which is meant a progressive wasting due to lesions in the spinal cord, as distinguished from the term *myopathies* or *dystrophies*, in which the primary disease is in the muscles themselves. Midway between these two types of muscular atrophies, both in the pathological lesions and clinical symptoms, is another, which will be described later as *progressive neural muscular atrophy*.

ETIOLOGY.—The direct cause of the progressive muscular atrophy of central origin is not known. Hereditary and family influences are, in adults, of much less importance in relation to this spinal type of progressive muscular atrophy than they are in the muscular atrophies of neural origin and of the dystrophies. The association of the disease, however, in the children of one family has been noted, and is a characteristic of the infantile form. Trauma, exposure to wet, cold, fright, and nervous shock are mentioned as etiological factors.

PATHOLOGY.—The pathological lesions consist primarily in a slow degeneration of the upper and lower neurons of the motor path, resulting in progressive atrophy of certain groups of muscles. The chief histological changes consist of an atrophy of the ganglion-cells of the anterior horns and an increase in the neuroglia. The lateral pyramidal tracts show degenerative changes, which, in some cases, have been traced to the motor cortex, and even to the cortical motor cells themselves. The direct cerebellar and ventro-lateral tracts are not involved. The muscles and the inter-muscular branches of the motor nerves show degenerative changes.

SYMPTOMS.—Indefinite pains suggesting those of chronic rheumatism may be the earliest symptoms. These are followed by a gradual loss of power and atrophy of the muscles. The muscles of the thumb, both in the thenar and hypothenar groups, and then the interossei and lumbricales are first affected, leaving depressions between the metacarpal bones. The wasting process then attacks the flexor and extensor muscles of the forearm, contractions set in, and the characteristic "claw-hand" develops. The deltoid is the first of the shoulder muscles to atrophy, and then follow in the typical cases the remaining muscles of the upper extremity, the muscles of the trunk, and finally those of the lower extremity. The trapezius and face muscles are among the first to be attacked. This order of progression is not always maintained. In rare cases the legs may begin to atrophy soon after the affection of the hand. Muscular contractures from the action of antagonistic muscles, fibrillary twitching in the unaffected muscles, numbness and coldness in the regions of atrophy, and diminution and even loss of muscular reaction to faradic and galvanic currents are generally present in advanced cases. The reaction of degeneration may be present in the very rapidly progressive lesions. The excitability of the nerve-trunks outlasts that of the muscles. The muscular weakness is proportionate and generally dependent upon the degree of atrophy. Sensation of heat, touch, and pain is not impaired.

In another type of cases, described by Charcot as *amyotrophic lateral sclerosis*, a spastic paralysis precedes the wasting, but so far as I know this condition does not occur in children.

In very rare cases in children the degenerative processes may affect the nuclei of the motor cranial nerves, both the upper and lower divisions of the motor tract being involved (Sachs). The symptoms in these cases bear a close resemblance to the progressive bulbar paralyses of adults, but are so rare that they need merely be referred to.

DIAGNOSIS.—The diagnosis is made on the slow, progressive wasting of muscles in the order described, on the gradual loss of power in the affected groups, on the fibrillary muscular contractions, on the diminution in the electrical reactions, on the absence of marked impairment of sensation, and, in the great majority of cases, on the absence of hereditary or family influences. The rarity of the disease in early life is also of some value in the diagnosis.

PROGNOSIS.—The disease is progressive and may last for many years. It is invariably fatal, although there may be periods in which there is an apparent arrest of the process, but which is only temporary.

TREATMENT.—The treatment is at best palliative and for the most part ineffective. Massage, electricity, anti-specific treatment if there is any evidence of a syphilitic taint, arsenic, and hypodermic injections of strychnine, as recommended by Powers, may be tried.

PROGRESSIVE NEURAL MUSCULAR ATROPHY.

Progressive neural muscular atrophy is also known as the *peroneal* or *leg type* of progressive muscular atrophy.

ETIOLOGY.—The disease is an hereditary or family affection, beginning in very early life in the cases reported by Sachs, and as late as twenty years in the case described by Charcot and Marie. Dubreuilh has reported a fatal and very typical case in a child in a family in which the mother and eleven children were similarly affected.

PATHOLOGY.—The pathological conditions found in Dubreuilh's case showed chronic degenerative changes in the peripheral nerves, without lesions in the gray substance of the spinal cord, with the exception of a very slight increase in the glia of the column of Goll. The motor nerves of the hands and feet were especially involved. The muscles showed atrophy of the fibres; some were degenerated and some were hypertrophied. The transverse striations were diminished and the nuclei increased. According to Sachs, these muscular changes more closely resemble those which occur in the primary dystrophies than those which are dependent on changes in the spinal ganglia, and the slight lesions in the eration may be present in the very rapidly progressive lesions. The excitability of the nerve-trunks outlasts that of the muscles. The muscular weakness is proportionate and generally dependent upon the degree of atrophy. Sensation of heat, touch, and pain is not impaired.

In another type of cases, described by Charcot as *amyotrophic lateral sclerosis*, a spastic paralysis precedes the wasting, but so far as I know this condition does not occur in children.

In very rare cases in children the degenerative processes may affect the nuclei of the motor cranial nerves, both the upper and lower divisions of the motor tract being involved (Sachs). The symptoms in these cases bear a close resemblance to the progressive bulbar paralyses of adults, but are so rare that they need merely be referred to.

DIAGNOSIS.—The diagnosis is made on the slow, progressive wasting of muscles in the order described, on the gradual loss of power in the affected groups, on the fibrillary muscular contractions, on the diminution in the electrical reactions, on the absence of marked impairment of sensation, and, in the great majority of cases, on the absence of hereditary or family influences. The rarity of the disease in early life is also of some value in the diagnosis.

PROGNOSIS.—The disease is progressive and may last for many years. It is invariably fatal, although there may be periods in which there is an apparent arrest of the process, but which is only temporary.

TREATMENT.—The treatment is at best palliative and for the most part ineffective. Massage, electricity, anti-specific treatment if there is any evidence of a syphilitic taint, arsenic, and hypodermic injections of strychnine, as recommended by Powers, may be tried.

PROGRESSIVE NEURAL MUSCULAR ATROPHY.

Progressive neural muscular atrophy is also known as the *peroneal* or *leg type* of progressive muscular atrophy.

ETIOLOGY.—The disease is an hereditary or family affection, beginning in very early life in the cases reported by Sachs, and as late as twenty years in the case described by Charcot and Marie. Dubreuilh has reported a fatal and very typical case in a child in a family in which the mother and eleven children were similarly affected.

PATHOLOGY.—The pathological conditions found in Dubreuilh's case showed chronic degenerative changes in the peripheral nerves, without lesions in the gray substance of the spinal cord, with the exception of a very slight increase in the glia of the column of Goll. The motor nerves of the hands and feet were especially involved. The muscles showed atrophy of the fibres; some were degenerated and some were hypertrophied. The transverse striations were diminished and the nuclei increased. According to Sachs, these muscular changes more closely resemble those which occur in the primary dystrophies than those which are dependent on changes in the spinal ganglia, and the slight lesions in the cord are probably secondary to those in the peripheral nerves. As this class of cases is intermediate between those of central origin and the primary dystrophies, he considers the neural origin of the disease a theory to be accepted with reserve until verified by other cases.

SYMPTOMS.—The peroneal muscles and the muscles of the feet are the first to be affected, atrophy sets in, contractions of antagonistic muscles follow, and end in the production of club-foot, either pes equinus or pes equino-varus. Eventually the entire leg may atrophy. The upper extremities and body are rarely affected until late in the disease, and the atrophy is not so marked as in the cases of central origin. In very rare instances the disease may begin in the hands. Fibrillary contractions and twitchings are present. The electrical reactions of both muscles and nerves are much diminished. The reflexes of the lower extremities are either lost or diminished. The sensations may be either normal or slightly altered; the sense of pain is sometimes increased. Muscular weakness proportionate to the atrophy and deformity is present, and gives rise to a waddling gait, and especially to difficulty in going up-stairs.

DIAGNOSIS.—The diagnosis of progressive neural muscular atrophy is to be made from several similar conditions.

From Progressive Muscular Atrophy of Central Origin.—This may be necessary in those cases in which atrophy of the lower extremities follows soon after the affection of the hands. The rarity of this condition in early life, the absence, as a rule, of hereditary influences, and the normal sensation are the only points in the differential diagnosis. Typical cases of either disease are readily distinguished by the onset and manner of progression, but in atypical cases the differential diagnosis may not be possible.

From Poliomyelitis.—In poliomyelitis the sudden onset, retrogressive character of the atrophy, the absence of hereditary influences, and the absence rather than diminution of knee-jerks serve to distinguish the disease from the slowly progressive hereditary paralysis which has just been described. The diagnosis from the subacute and chronic forms of poliomyelitis is often difficult until the progressive character of the disease becomes apparent.

From Hereditary Ataxia.—The absence of ataxia, the abnormal electrical reactions, and the persistence of the reflexes in certain cases of progressive neural muscular atrophy render the diagnosis from hereditary ataxia clear in most cases.

From Chronic Multiple Neuritis.—In the muscular atrophy dependent upon neuritis, the greater prominence of pain and tenderness, the absence of hereditary or family influences, and the extreme rarity of the development of double club-foot generally enables us to distinguish neuritis from progressive neural muscular atrophy.

Déjerine and Soltas have described a family disease of rare occurrence beginning early in life and resembling in many respects a progressive neural muscular atrophy. It is characterized by the symptoms of tabes dorsalis combined with progressive muscular atrophy involving the face and lips and by an interstitial hypertrophic neuritis extending into the dorsal columns of the cord. The condition is sometimes called *progressive interstitial hypertrophic neuritis of infants.*

PROGNOSIS.—The duration of the disease is very chronic, but there may be periods of arrest in its development.

TREATMENT.—The disease is incurable. Much can be done by orthopædic surgery for the relief of deformities produced by the contractions. Massage and electricity are the main indications for treatment.

PROGRESSIVE MUSCULAR DYSTROPHIES.

CLASSIFICATION.—The group of diseases known as *primary muscular dystrophies*, or *primary myopathies*, are represented by several forms, which differ from progressive muscular atrophy of central origin (Aran-Duchenne type) in that the lesions are primary in the muscles and are not dependent upon degenerative changes in the spinal cord.

For the sake of convenience Erb has divided the muscular dystrophies into two large groups, which are still further subdivided into types dependent partly on pathological and partly upon clinical differences. This classification may be expressed as follows:

I. Progressive Muscular Dystrophy of Infancy.
    A. Hypertrophic Form, in which the muscles are increased in size, and eventually become atrophic.
        a. With real hypertrophy of the muscle fibres.
        b. With false hypertrophy, the muscular fibres having undergone lipomatosis.
    B. Atrophic Form, in which there is no stage of hypertrophy.
        a. The Landouzy-Déjerine type, in which there is primary involvement of the face.
        b. An atrophic form without involvement of the facial muscles.
II. Progressive Muscular Atrophy of Youth and Adult Life. This is also known as *Erb's juvenile* form.

All these forms have certain points in common. There is a distinct hereditary influence or family association. The electrical reactions are diminished in all, but none show a typical reaction of degeneration. The reflexes diminish proportionately to the degree of muscular atrophy. As Sachs has pointed out, the chief distinction therefore between these clinical types are in reference to the distribution of the atrophy or hypertrophy.

ETIOLOGY OF THE MUSCULAR DYSTROPHIES.—Heredity or association of cases in one family are the only etiological factors of any importance, so far as is known. This peculiarity serves as the most important point in the diagnosis of the dystrophies from progressive central muscular atrophy, almost all cases of which, except Erb's infantile form, are free from hereditary influences. The disease is usually transmitted through the mother, who may not herself be affected, may run through as many as five generations, and generally attacks the males. The disease, as a rule, begins before puberty, rarely after the twentieth year.

PATHOLOGY.—The chief histological changes in the muscular dystrophies consist in hypertrophy and atrophy of the muscular fibres, with proliferation of the nuclei, vacuolization and segmentation of the fibres. The hypertrophy of the muscle fibres appears to be an early process in all forms of muscular dystrophies, and is associated with lipomatosis and changes in the connective tissue. Atrophy of the muscle fibres is a later stage of the disease. No sharp distinction histologically can be drawn between the muscular lesions of the spinal amyotrophies and the muscular dystrophies, as hypertrophy of the muscle fibres may rarely occur in the spinal amyotrophies (Sachs). The dystrophies are, however, not associated with lesions of the central nervous system.

SYMPTOMS.—**Pseudo-Hypertrophic Forms.**—The earliest symptoms noticed are weakness of the muscles, a shuffling, clumsy gait, and awkwardness in the ordinary motions, especially in rising from the floor or climbing stairs. These symptoms generally precede any noticeable enlargement of the muscles. Gradually, however, the muscles of the calves increase in size and are hard and firm. The thighs are rarely hypertrophied. Atrophy of the thighs and of the deep muscles of the back, shoulder, and scapulæ sets in. Late in the disease the atrophic process becomes general, affecting even the hypertrophied calves. The muscles of the face are not involved until in the very last stages.

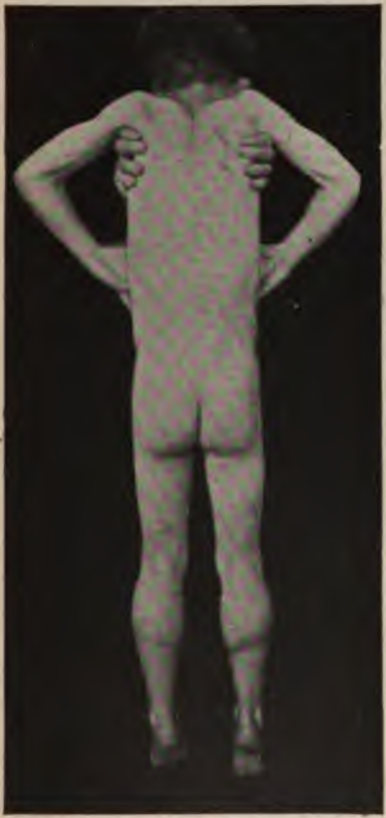
Children who are affected with this form of dystrophy learn to walk late, and assist themselves by leaning on the furniture or other objects in their path. When the child is placed on the floor on its back it has difficulty in getting up. It has to turn over on its face first, and then to aid the weakened muscles of the legs and trunk by means of the hands and arms, climbing up, as it were, upon itself by placing the hands upon the knees and then farther and farther up the thighs. Fibrillary contractions do not occur. The knee-jerks in some cases disappear as the disease advances. Sensation, as a rule, is normal. There is seldom any disturbance of the bladder or rectum.

In the later stages of the disease contractions of the muscles occur, and in this way permanent distortions of the joints may result. The most common deformities are talipes equinus and flexion of the knees and hips.

Lateral curvature occurs, or a permanent flexion of the spine from weakness of the erector spinæ muscles may result so that the child sits bowed forward, or when kneeling on the hands and feet there is a saddle-shaped depression of the back. The electrical reactions show only quantitative changes. The reaction of degeneration is never present.

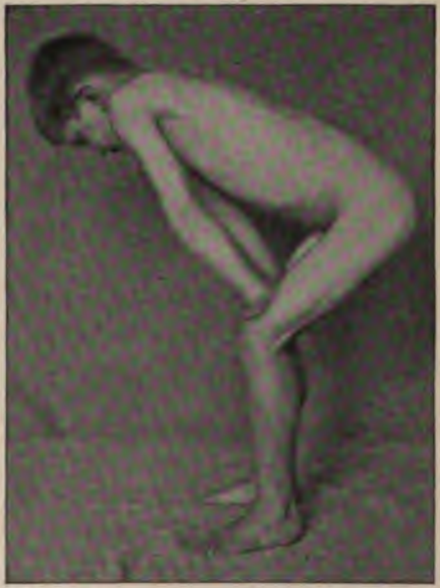
Atrophic Form.—This form is known as the *Landouzy-Déjerine type.* The muscles of the face and shoulder are primarily affected; there is no hypertrophy except in very rare instances. The muscles of the forearm, hands, legs, and back remain unaffected. Westphal has described a case in which the face muscles were involved in an otherwise typical case of pseudo-hypertrophy. Other forms occur without involvement of the facial muscles. The Landouzy-Déjerine type, therefore, is not absolutely distinctive. I have reported a case of this affection in the first edition of this work.

FIG. 203.



Pseudo-hypertrophic muscular paralysis, showing enlarged calves.

FIG. 204.



Pseudo-hypertrophic muscular paralysis, showing position assumed in rising from the floor.

Erb's Juvenile Form.—The shoulder muscles are primarly affected. The disease begins in late childhood or early youth, and is characterized by progressive atrophy of the muscles of the shoulder, upper arm, pelvis, thigh, and back, the forearm and legs remaining normal. True or pseudo-hypertrophy may be associated with the atrophy in certain groups of muscles, notably in the deltoids, supra-spinati and infra-spinati muscles.

Diagnosis.—The differential diagnosis of the different forms of progressive muscular dystrophies present many difficulties, and depend primarily upon the distribution of the lesions, as in all other respects they may be practically the same.

The group differs from the spinal or central form, however, in the hereditary character of the affection, in the primary appearance and the distribution of the lesions, in the absence of fibrillary contractions, in the absence of marked electrical changes, and in their greater frequency in early life.

The chief point in the diagnosis from progressive neural muscular atrophy is in the peculiar distribution of the atrophy in the latter affection to the muscles of the feet and to the peroneal group.

Prognosis.—All forms of the muscular dystrophies are slowly progressive and are incurable. The pseudo-hypertrophic type may render the patient a helpless cripple in two or three years. The other forms may last for ten, twenty, or thirty years, but the patient is very susceptible to intercurrent diseases.

Treatment.—The treatment is very limited and does not differ from that of progressive central muscular atrophy described on page 991.

Fig. 203 illustrates the hypertrophy of the calves which occurs in the pseudo-hypertrophic form of progressive muscular atrophy. Fig. 204 represents a child with the same condition in the characteristic act of "climbing up on himself."

DIVISION XVII.

UNCLASSIFIED DISEASES.

HÆMOPHILIA.

In contradistinction to the hemorrhages of infectious origin which occur in the early weeks of life is that class of hemorrhages which can be classed under the term *hæmophilia*.

Hæmophilia simply means a morbid condition characterized by a tendency to bleed spontaneously or from any insignificant wound. Individuals who are liable to bleed in this way are designated as having a *hemorrhagic diathesis*. The disease is not especially common in the early weeks of life, and usually occurs at a later period of development. It begins to be more frequent towards the end of the first year, and is apparently well established in the second year and later in childhood. It does not have a self-limited course, as is the case with the other form of hemorrhage. It is not infectious, and is not accompanied by fever. It may be for many years masked, and then may become manifest from some trivial cause, such as the extraction of a tooth. It is a dangerous disease, and death is very liable to occur from inability to control the hemorrhage. The condition is hereditary, being transmitted through the females to the males, but seldom occurring in the females, the proportion being one to eleven or thirteen (Osler).

In addition to the hemorrhage which occurs in various parts in hæmophilia König describes a condition of the joints which for a long time was supposed to be of gouty origin. This affection of the joints, like other hæmophilic hemorrhages, occurs most commonly in boys and young men. The onset of the attack is sudden, and often without any history of trauma. The effusion into the joint is found to be of pure blood, and the quantity may be large. This effusion may be entirely absorbed spontaneously, or may occur later in the same joint or elsewhere. Shaw reports a case in which the recurring effusion into and about the joints was regularly preceded by a definite prodromal discomfort in the joint. After repeated attacks, chronic joint changes may ensue. There is no tendency to suppuration. The effusion sometimes occurs about, rather than in the joint. The disease is liable to be mistaken for, and is to be differentiated from, tumor albus and tuberculosis. Careful attention to personal and family history is the only safeguard in making a diagnosis of this disease, although we should always bear in mind the possibility of its presence in case of the affection of a joint of sudden onset in a young male and without history of severe traumatism.

TREATMENT.—There is no treatment which has been found successful in these cases beyond the active local employment of styptics and compression.

PURPURA.

Purpura is a term applied to certain conditions in which there are hemorrhages into the skin or mucous membranes. These hemorrhages may be of various sizes. When small, they are called petechiæ; when larger, they are called ecchymoses. There is no proof that purpura is a disease of the blood. Its etiology is very obscure, and although this condition has been divided into various forms, such as purpura simplex and purpura hemorrhagica, it is doubtful whether these are not all microbic in their origin and simply represent different degrees of infection.

In the more simple forms of purpura the hemorrhages are only in the skin, while in the more severe affection the mucous membranes of the mouth and gastro-enteric tract are usually involved.

Not only does purpura occur in what may be called primary forms, but this purpuric condition may also be secondary to a number of diseases, especially those of an exhausting nature. Thus, I have seen it in the more severe and later stages of infantile atrophy, in which the hemorrhages may cover almost the entire front of the body. It may also be a symptom in the more severe cases of measles, scarlet fever, varicella, variola, diphtheria, and in the course of a prolonged rheumatic attack.

In an infant who died of infantile atrophy at the Infants' Hospital the skin of the extremities showed numerous ecchymoses of various sizes and of a dark red and purple color. On the thorax on both sides above the nipples were two large ecchymoses, and there were smaller ecchymoses all over the rest of the trunk. On post-mortem examination nothing abnormal was found except a slight atelectasis of the lower lobes of both lungs, with pleuritic adhesions at the base of the right lung and slight granular degeneration of the heart, liver, and kidneys, with hyperplasia of the mesenteric lymph-nodes.

Purpura Simplex.—In the simple forms of purpura the disease in children is often mild, and is accompanied by a loss of appetite, slight anæmia, a slight degree of fever, and the appearance of petechiæ in various parts of the skin. The prognosis is good, and the duration of these attacks is usually from one to two weeks. They are at times associated with pains located in various places.

Purpura Rheumatica.—The form which has been called purpura rheumatica (*peliosis rheumatica; Schönlein's disease*) probably has no connection with rheumatism beyond the possibility of their both being microbic, and merely simulates rheumatic arthritis from the fact that it affects the joints. The diagnosis is made by the characteristic association of multiple arthritis with purpura and urticaria.

Henoch's Purpura.—Closely simulating and probably representing purpura rheumatica, except that the gastro-enteric symptoms are more prominent, is a form which has been called Henoch's purpura. It occurs especially in children between the ages of three and nine years. Its direct cause is not known, although it usually occurs among children who have had bad hygienic surroundings and have been ill cared for.

SYMPTOMS.—The symptoms are more or less malaise, and pains not especially localized, but chiefly occurring in the extremities and back, sometimes accompanied by slight œdema of the part affected. These early symptoms of pain occur in one or more joints, usually on the outer sides, and sometimes there are swelling and redness simulating articular rheumatism. In this stage there may be a sudden rise of temperature. Accompanying these symptoms there may be a few purpuric spots, but, as a rule, there is a period of several days between the appearance of the pains in the joints and the purpuric appearances on the skin. The purpuric spots may coalesce, and thus form ecchymoses of various sizes and colors. They are very apt to begin on the lower leg and spread up to the thighs, genitals, and body. Somewhat later intestinal symptoms develop. While the purpura is spreading there is severe colic, and the pain is very intractable to treatment. The abdomen is retracted and tender. There is obstinate vomiting. The pulse is weak, and the face has an anxious expression. There is more or less diarrhœa, which usually occurs at the end of an attack of colic. The colic and vomiting sometimes last for one or two days. There may be a little blood in the vomitus and in the movements. The vomiting then diminishes, the colic ceases, and later the diarrhœa stops, the pain in the joints passes away, the purpuric spots gradually fade and disappear, and the child, although left in an exhausted condition, is otherwise well.

There are very apt to be relapses, which may appear within a few days or not for several weeks.

These are the symptoms of a typical case; but there are many variations. As a rule, the younger the child the more typical is the case. Sometimes the purpuric spots closely simulate urticaria. They may occur, although rarely, in the mouth. They sometimes simulate the lesions of erythema nodosum. The attacks of colic have a paroxysmal character. There may be swelling of the joints.

The disease is rarely fatal unless it is complicated by some such disease as nephritis or endocarditis. The treatment is purely symptomatic.

Purpura Hæmorrhagica.—The most severe form of purpura which occurs is that which is called purpura hæmorrhagica (*morbus maculosus Werlhofii*). The hemorrhages in this form are from the mucous membranes as well as into the skin. The disease begins with debility. A few days later purpuric spots appear on the skin, and subsequently hæmarecognition of the general lymphatic enlargement and the exclusion of other causes, such as tuberculosis and leukæmia.

PROGNOSIS.—The special tendency to sudden death which exists in this condition must be borne in mind. The vitality of the child is distinctly lowered, and it is especially susceptible to the toxic actions of the acute infectious diseases. At the same time there is a tendency to outgrow this condition as puberty approaches, so that with careful management the prognosis may become favorable.

TREATMENT.—The treatment is essentially symptomatic, and consists chiefly in the regulation of the food and hygiene of the child.

MUSCULAR RHEUMATISM (Myalgia).

Muscular rheumatism is an affection characterized by pain and stiffness in certain groups of muscles. It may be acute or chronic.

ETIOLOGY.—The disease is usually seen in children between the ages of five and fifteen, and generally follows exposure to cold or dampness and to bad hygienic surroundings. It has been held by Leube, Peltessohn, and others to be due to infectious micro-organisms, but there is as yet no proof of this.

SYMPTOMS.—Pain on movement and tenderness and stiffness of certain muscles are the typical symptoms. Fever is rarely present. The pain is at times quite severe, slight movements of the muscles bringing on sharp exacerbations. Certain names have been given according to the group of muscles affected, such as *cervicodynia*, or *acute torticollis*, when the process is located in the muscles of the neck; *pleurodynia* when in the intercostal muscles; *lumbago* when in the lumbar muscles; *cephalodynia* when in the muscles of the head; *scapulodynia* and *omodynia* when in the muscles of the shoulder.

The disease may be acute or chronic, the acute cases recovering in a few days. The chronic cases may persist for weeks, often leading to increase in the connective tissue, producing muscular stiffness and contractures.

DIAGNOSIS.—The diagnosis is to be made from pleurisy by the absence of physical signs and by the pain and muscular tenderness. It is distinguished also from neuritis, in which the tenderness is along the course of the nerves and in which massage is not efficient.

TREATMENT.—Drugs are of less use than hot air and massage. Heat may be applied by means of a hot flat-iron rubbed on the affected part protected by a piece of thick flannel. Massage is especially to be recommended. Electricity is of service in chronic cases. Phenacetine and the salicylates are the drugs which are indicated; they should be used with caution if they seem to benefit the especial case, and should not be employed as a routine treatment. The lithia waters should be given freely.

The following case is illustrative of acute torticollis dependent upon acute muscular rheumatism:

recognition of the general lymphatic enlargement and the exclusion of other causes, such as tuberculosis and leukæmia.

Prognosis.—The special tendency to sudden death which exists in this condition must be borne in mind. The vitality of the child is distinctly lowered, and it is especially susceptible to the toxic actions of the acute infectious diseases. At the same time there is a tendency to outgrow this condition as puberty approaches, so that with careful management the prognosis may become favorable.

Treatment.—The treatment is essentially symptomatic, and consists chiefly in the regulation of the food and hygiene of the child.

MUSCULAR RHEUMATISM (Myalgia).

Muscular rheumatism is an affection characterized by pain and stiffness in certain groups of muscles. It may be acute or chronic.

Etiology.—The disease is usually seen in children between the ages of five and fifteen, and generally follows exposure to cold or dampness and to bad hygienic surroundings. It has been held by Leube, Peltessohn, and others to be due to infectious micro-organisms, but there is as yet no proof of this.

Symptoms.—Pain on movement and tenderness and stiffness of certain muscles are the typical symptoms. Fever is rarely present. The pain is at times quite severe, slight movements of the muscles bringing on sharp exacerbations. Certain names have been given according to the group of muscles affected, such as *cervicodynia*, or *acute torticollis*, when the process is located in the muscles of the neck; *pleurodynia* when in the intercostal muscles; *lumbago* when in the lumbar muscles; *cephalodynia* when in the muscles of the head; *scapulodynia* and *omodynia* when in the muscles of the shoulder.

The disease may be acute or chronic, the acute cases recovering in a few days. The chronic cases may persist for weeks, often leading to increase in the connective tissue, producing muscular stiffness and contractures.

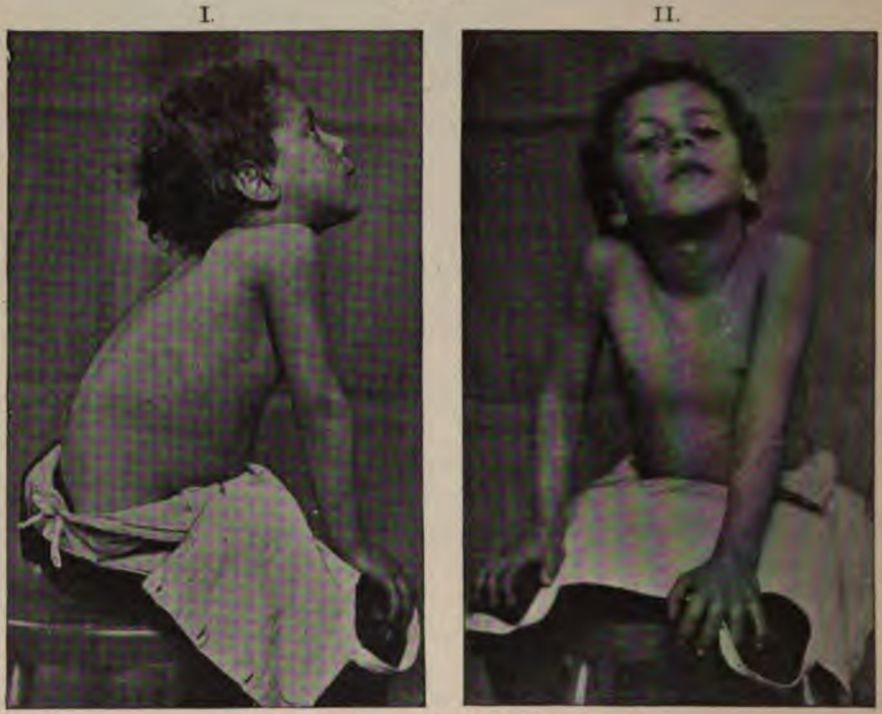
Diagnosis.—The diagnosis is to be made from pleurisy by the absence of physical signs and by the pain and muscular tenderness. It is distinguished also from neuritis, in which the tenderness is along the course of the nerves and in which massage is not efficient.

Treatment.—Drugs are of less use than hot air and massage. Heat may be applied by means of a hot flat-iron rubbed on the affected part protected by a piece of thick flannel. Massage is especially to be recommended. Electricity is of service in chronic cases. Phenacetine and the salicylates are the drugs which are indicated; they should be used with caution if they seem to benefit the especial case, and should not be employed as a routine treatment. The lithia waters should be given freely.

The following case is illustrative of acute torticollis dependent upon acute muscular rheumatism:

The boy was five years old. Since he was three years old he had been subject to attacks of torticollis, apparently of rheumatic origin. He was brought into the hospital in one of these attacks. His temperature was somewhat raised, and he had a slight loss of appetite, but otherwise was perfectly well, and did not suffer any pain except when his neck was touched. The head was drawn rigidly back. These paroxysmal attacks usually lasted two or three days, when they passed off as suddenly as they came. The last attack which he had was one year before.

Fig. 205.



Acute rheumatic torticollis. Fifth day of attack.

On the following day the stiffness and the pain in the neck passed off, and the head resumed its normal position. Various drugs had been given in these attacks, but none with any especial benefit except salicylate of sodium, which seemed to control the pain.

I have also had under my care a little boy, about four years of age, who was attacked with fever, pain in the region of the spine, and spasm along the entire length of the spinal column. There was no pain or tenderness anywhere except over the vertebral column, and these symptoms were not so marked in the cervical region as lower down. The child had no mental disturbance, but for a number of days was in a condition of continued opisthotonos from the hips upward, so that he had to be kept in a reclining chair with pillows under his arched back. The normal functions of the bladder and intestine were not interfered with. The pulse was rapid, the temperature was moderately raised, and the respirations were normal. The appetite was lessened. He remained in this condition for about a week, when the spasm of the back began to disappear. The muscles relaxed for a short time and then stiffened again. Finally complete relaxation took place, and the child recovered entirely. The attack was acute in its onset, and did not follow any injury. The treatment was with bromide of potassium, 0.3 gramme (5 grains) three or four times in the twenty-four hours.

ARTHRITIS DEFORMANS.

Arthritis deformans, while generally a disease of adult life, may occur in children, usually after the age of six, and may present the same characteristics as in later life.

There is, however, in young children a special variety of the disease, which has been described by Still. This form generally occurs before the second dentition, and is more frequent in girls than in boys. There is a general enlargement of the joints associated with swelling of the spleen and lymph-nodes. The onset is sometimes acute, with fever and even chills. The joints gradually become stiffened and enlarged, due to a proliferation of the soft tissues rather than of a bony enlargement. The limitation of motion and muscular wasting about the joints is often pronounced, but there is no bony grating. There may be profuse sweating. The blood shows only a secondary anæmia.

PROGNOSIS.—The disease is incurable, and progresses slowly for many years. At times an arrest of the disease occurs.

TREATMENT.—The treatment by drugs is useless, except so far as to control the symptoms. Arsenic and iron are useful for the anæmia. Food and fresh air, hydrotherapy, massage, and hot-air are the principal indications for treatment.

CHRONIC RHEUMATISM.

ETIOLOGY.—Chronic rheumatism may occur in children as a result of acute rheumatic fever, but is more likely to come on insidiously in connection with unhealthy surroundings, poor food, and undue exposure to cold and dampness.

PATHOLOGY.—The pathological lesions are represented by injection of the synovial membranes, thickening of the capsules and ligaments of the joints and of the neighboring tendon sheaths, and erosions of the cartilages if the process has been of long duration.

SYMPTOMS.—Pain and stiffness in the joints with a susceptibility to exacerbations, during which swelling and tenderness without redness set in, are the chief symptoms. Ankylosis of the joints may occur eventually. Debility and anæmia appear as secondary conditions. The disease is usually persistent and slowly progressive, but does not directly shorten life.

TREATMENT.—Avoidance of cold weather and dampness is desirable. General hygiene, good food, massage, and the treatment with hot air are of great benefit. Many cases will improve under treatment at various hot springs.

DIABETES INSIPIDUS.

Diabetes insipidus is a disease characterized by the passage of large quantities of urine of very low specific gravity, free from sugar and other abnormal elements, and often with a considerable increase in the total amount of solids.

ETIOLOGY.—The cause of the disease is not known. It is probably in the nature of a neurosis, causing a vasomotor disturbance of the renal vessels with continuous congestion of the kidney. In a series of eighty-five cases collected by Strauss, nine were under five years and twelve between five and ten years. In seventy cases reported by Roberts, twenty-two occurred before ten years. It may be congenital or begin in infancy, but is an exceedingly rare disease. In certain cases there is a strong hereditary tendency. It has been recorded in the members of four generations of one family (Weil).

PATHOLOGY.—Enlargement and congestion of the kidneys, hypertrophy of the bladder, dilatation of the ureters and pelves of the kidney, and inconstant lesions of the nervous system have been noted.

SYMPTOMS.—The symptoms are directed especially to the urine, which is passed in very large quantities; in one case of Trousseau's, in an adult, amounting to twenty-eight quarts a day. There is a very low specific gravity, varying from 1001 to 1005 or 1007. Glucose is never present, but inosite (muscle-sugar) has at times been noted. Albumin and casts are of only rare occurrence. The total solids are normal or in many cases greatly increased.

Intense thirst, a dry skin, disturbance in the surface circulation, and general nervous symptoms are present, but emaciation is not apt to occur as in diabetes mellitus. The appetite is good. Often the general health is not interfered with, and the disease may last for a period of years.

DIAGNOSIS.—The condition is to be distinguished from nervous or hysterical polyuria, which is of temporary duration and is associated with other hysterical symptoms.

It is differentiated from diabetes mellitus by the absence in the urine of glucose or grape-sugar.

Chronic interstitial nephritis may be excluded from the diagnosis by the presence of albumin and casts and the low percentage of solids eliminated, together with the clinical symptom of chronic nephritis.

PROGNOSIS.—Occasionally spontaneous recovery may take place, but oftener the disease is likely to remain chronic and the child to die of some intercurrent affection.

TREATMENT.—A nitrogenous diet with small amounts of carbohydrates and only a moderate restriction of fluids are indicated. Drugs do not control the disease, although many have been recommended, the most prominent of these being belladonna in increasing doses up to its physiological limits and opium, continued for many months. Arsenic and the bromides may also be tried, but their beneficial action is doubtful.

DIABETES MELLITUS.

Diabetes mellitus is a disease characterized by the excretion in the urine of glucose or grape-sugar in considerable quantities and for a long period. It is sometimes acute, but is generally chronic. It is to be distinguished from the condition known as temporary *glycosuria*, in which sugar is present in the urine for a short time only, as after certain acute infectious diseases, injuries to the head, or from absorption of some toxic substance. There is still another variety of glycosuria which is spoken of as *temporary alimentary glycosuria*. In this form sugar appears in the urine after the ingestion of large quantities of saccharine foods, and disappears at once upon reducing the sugar in the diet.

ETIOLOGY.—Diabetes mellitus may occur in infancy or at any period of childhood. It is very common among the Jews, and is infrequent among the negroes.

It occurs more frequently in girls than in boys. In Stern's analysis of one hundred and seventeen cases in children, six were under one year of age.

In many cases an hereditary influence seems to be a factor in the etiology; in others a severe nervous shock, such as may come from sudden fright or great grief, may be an exciting cause. Trousseau states that the children of tubercular parents are particularly susceptible to diabetes. Injuries to the head, especially if the floor of the fourth ventricle is involved, may cause the appearance of sugar in the urine. Occasionally temporary glycosuria may arise from the administration of antitoxin, and it may appear shortly before death in cases of chronic diffuse and subacute glomerular nephritis.

Pancreatic diabetes follows the removal of the pancreas in dogs, and lesions in the pancreas are found in about half of the cases of diabetes mellitus; the appearance of sugar in the urine in these cases is supposed to be due to the absence of the glycolytic ferment in the pancreatic juice.

Phloridzin and a few other substances may cause diabetes mellitus by destroying the power of the epithelium to keep back the sugar.

Faulty metabolism and defective assimilation of the glucose probably underlie most cases of diabetes, but much is to be learned as to the real nature of the disease.

PATHOLOGY.—Lesions in the medulla from tumors or scleroses have been found, and glycogen has been found in the spinal cord. There may be a secondary multiple neuritis. The blood may contain as much as 0.4 per cent. of sugar instead of the normal 0.15 per cent. The plasma is fatty and the polynuclear leucocytes contain glycogen. Tuberculosis of the lungs is not uncommon, and may follow an acute broncho- or pneumococcus lobar pneumonia. The liver is enlarged and fatty. Lesions of the pancreas, generally atrophy, are present in nearly fifty per cent. of the cases. The kidneys usually show evidence of diffuse nephritis with fatty degeneration.

SYMPTOMS.—The symptoms in the temporary form of diabetes may be slight, and the sugar is discovered only in the routine examination of the urine. There may be an increased flow of urine and a craving for sweet articles of diet. The appetite is good. The thirst may be increased. Indigestion is not uncommon.

In true diabetes the symptoms are much more severe. Great thirst, excessive appetite, and polyuria are the striking symptoms. There is progressive and extreme emaciation, the skin is harsh and dry; sweating is rare. Constipation is usually present. The flow of saliva is abundant. The genitals are at times irritated by the urine.

Urine.—The quantity of urine is greatly increased, generally averaging between 3000 to 5000 c.c. (100 to 166 ounces), although it may be much more or be less than normal. The urine is pale, strongly acid, and of high specific gravity, the degree depending upon the amount of sugar present. The solids are relatively diminished but absolutely increased in amount. Glucose or sugar is always present, and varies from a mere trace to 8, 10, or 12 per cent., and the total quantity in twenty-four hours varies from 20 to 60 grammes (⅔ to 2 ounces), often amounting to several hundred grammes. There is usually the slightest possible trace of albumin and a few casts, indicative of a mild, active hyperæmia of the kidneys.

Diabetic Coma.—Diabetes mellitus frequently ends in coma, this presumably being brought about by some toxic substance in the blood. This complication is especially common in children. It is at times precipitated by sudden changes in diet, especially when the patient is put upon a rigid, nitrogenous diet, but it may also follow many other conditions, such as intercurrent acute diseases and operations.

Acetone and diacetic acid are found in the urine in advanced cases of diabetes, and are always of serious prognosis, as they indicate a threatened state of coma, but they may be present for months before any symptoms of coma appear, and may even disappear as the patient improves under treatment. Acetone and diacetic acid are probably rarely, if ever, present in cases of temporary glycosuria.

The advent of coma may be suspected by the appearance of mental apathy, nausea, vomiting, and epigastric pain. The breath has a peculiar odor of acetone. Convulsions rarely occur, which is a point of considerable value in excluding a coma due to uræmia. *Bremer's blood-test* with methylene-blue or congo-red will also aid in determining the nature of the coma, as a positive reaction will almost always be obtained, even in the early stages of diabetes mellitus.

COMPLICATIONS.—Boils, furuncles, eczema, and prurigo are common complications. Gangrene may occur. Pneumococcus lobar pneumonia, broncho-pneumonia, and especially tuberculosis of the lungs, are likely to occur and prove fatal.

Peripheral neuritis with pain and paralysis may occur, sometimes with symptoms suggestive of tabes. The disease may be manifested by paralysis, either the arms or legs being affected, the form being diplegic. Absence of the knee-jerk is not uncommon, and is probably due to neuritis. Mental depression or extreme restlessness or nervousness often develops.

Cataract, retinitis, sudden amaurosis, ocular paralysis, and atrophy of the optic nerves occasionally occur. Otitis media or a mastoiditis may appear suddenly. Œdema is a common symptom, and does not always depend on disease of the heart or kidney.

Diagnosis.—It is important to distinguish between diabetes mellitus and temporary glycosuria. This is sometimes difficult. In the former, however, sugar is present in every specimen of urine passed, irrespective of the time of day. In temporary alimentary glycosuria the urine in the early morning, after ten or twelve hours of fasting, is generally free from sugar, or only traces are present, whereas after a meal considerable quantities of sugar are eliminated. A rigid diet, free from carbohydrates, will also help to make the distinction clear, as the quantity of sugar in true diabetes mellitus is reduced only with difficulty and often cannot be markedly influenced.

From other forms of polyuria the diagnosis is made clear by the presence of sugar as determined by Fehling's test.

Bremer's blood-test with congo-red is a valuable aid in differentiating true diabetes mellitus from phloridzin diabetes.

Prognosis.—The disease runs a much more rapid course in children than in adults. In Stern's one hundred and seventeen cases the shortest duration was two days. In seven cases death took place within a month. He mentions one case of a child whose urine contained sugar at birth, and who recovered in eight months. In children the disease is almost universally fatal, but C. Sterns, in a series of seventy-seven cases in children, found that fourteen recovered, and in one hundred and eight cases, also children, reported by Wegeli, only sixty-four terminated fatally.

The younger the child the more grave is the prognosis.

Treatment.—The treatment of diabetes mellitus consists practically in the regulation of the diet, the endeavor being to limit the ingestion of carbohydrates. The diet should consist, so far as possible, of meat, fish, eggs, green vegetables, salads, and nuts. Too great a quantity of meat should be avoided, as excessive nitrogenous food as well as carbohydrates will increase the percentage of sugar in the urine.

It is well to test the patient's capacity to assimilate sugar by quantitating the sugar passed in twenty-four hours when on ordinary diet, and then to institute a strict diet for several days, the sugar being estimated daily. The results will give us indications as to whether to push the restricted diet or to allow more freedom in the choice of foods. When coma is threatened, or when the reaction for diacetic acid with ferric chloride is positive, carbohydrates should be added to the diet for a time.

If under a strict diet the sugar disappears, the diet can gradually and cautiously be increased, but at intervals of a few months diabetics should return to their restricted diet for a few weeks as a matter of precaution and to increase their power of assimilation of the carbohydrates.

The following articles represent as varied a diet list for diabetics as is compatible with conservative treatment: oxtail soup, bouillon, and clear soups made from meats; lemonade, chocolate, and cocoa, with saccharin for sweetening; soda-water and charged waters.

Fresh fish of all sorts, with oysters in season, poultry, game, eggs, butter, buttermilk, and cream cheese, lettuce, tomatoes, spinach, chicory, sorrel, radishes, asparagus, watercress, cucumbers, celery, and endives are permissible.

Of the fruits, lemons, oranges, currants, plums, cherries, pears, apples, melons, raspberries, and strawberries may be taken in moderation if a strict diet is not being adhered to. Nuts may be given if they do not disturb digestion.

Among the articles especially to be avoided are thick soups, liver, all forms of bread, and all dishes prepared with flour or sugar. Vegetables growing under ground are to be avoided. Beers, wines, and alcoholic drinks should not be used.

Most of the gluten breads and diabetic flours in the markets are adulterated with flour, and cannot be relied upon.

A mixture of practically sugar-free milk can be obtained at the various milk-laboratories, and will probably in the future prove to be of much use in the dietetic treatment of diabetes, especially in infancy.

But little is to be expected in the treatment of diabetes from the use of drugs, except so far as to allay symptoms. Opium, usually given in the form of codeia, is quite generally used, but I have not myself seen striking results from its administration.

INDEX.

A.

Abdomen, anatomy and relations of the, 57; at term, 33
Abscess, cerebral, 949; of the liver, 836; of the lung, 707; peritonsillar, 654; psoas, 429; retropharyngeal, 656
Acardia, 721
Acarus scabiei, 355
Acetone in diabetes mellitus, 1006
Achondroplasia, 342
Achorion Schoenleinii, 360
Acidophilic, 875
Acquired disease, definition of, 19
Acute circumscribed œdema, 379
  fatty degeneration of the new-born, 312
  fermental diarrhœa, 805
  infectious osteomyelitis, 51
  miliary tuberculosis of the lungs, 393
  nervous diarrhœa, 796
  nervous vomiting, 774
  torticollis, 1001
  yellow atrophy of the liver, 835
Addison's disease, 908
Adenitis, simple acute, 897; simple chronic, 898
Adenoid growths, 645
Adolescence, albuminuria of, 848
Adrenal gland, diseases of the, 908
Æstivo-autumnal parasite, 483, 484
Age, determination of, in premature infants, 257
Agenesis corticalis, 931, 951
Albuminuria of adolescence, 848
  physiological, 847
Albumin water, preparation of, 791
Alopecia areata, 361; in syphilis, 525
Amaurosis in scarlet fever, 573
Amnesia, temporary, 937
Amœbic ileo-colitis, 501, 820
Amyloid degeneration of the heart, 734
  infiltration of the kidney, 859; of the liver, 837
Amyotrophic lateral sclerosis, 991
Amyotrophies, 990
Anæmia, primary or pernicious, 890; pseudoleukæmic, 888; secondary, 894
Anatomy of infants and children *vide* Infant at term, and Normal development; topographical, 88
Anencephalia, 290
Aneurism, 720; intra-cranial, 978
Angio-neurotic œdema, 379, 943
Animal parasites, 829; ascaris lumbricoides, 831; oxyuris vermicularis, 829; tænia saginata or mediocanellata, 832; taenia solium, 832
Antitoxin, dose of, 471; immunizing dose of, 469; technique of injection of, 473; in treatment of diphtheria, 471
Anuria, 847
Anus, imperforate, 307; in syphilis, 827
Anopheles, genus, in malaria, 481; quadrimaculatus, 482
Aorta, transpositions of, 721
Aortic insufficiency, 743; stenosis, 744
Aortic orifice, lesions of the, in congenital disease, 726
Aphasia, organic, 938; temporary, 937
Appendicitis, 815; catarrhal, 816; chronic, 818; diagnosis, 819; etiology, 815; obliterative, 818; pathology, 815; prognosis, 819; simple, 816; symptoms, 816; treatment, 819; ulcerative, 816; with general suppurative peritonitis, 817; with peri-appendicular peritonitis, 817; with septicæmia, 818
Aran-Duchenne type of progressive muscular atrophy, 989
Arrested psychical development, 938
Arteries, transposition of the large, 726
Arteritis umbilicalis, 301
Arthritis of infants, 511; deformans, 1003
Ascaris lumbricoides, 831
Ascending colon, relations of, 69
Ascites. (*Vide* symptoms of the especial diseases.)
Asphyxia in the new-born, 312
Aspiration pneumonia, 691
Asthma, bronchial, 673; dyspepticum, 946; false, 674
Ataxia, hereditary, 988; locomotor, 989
Atelectasis, 705; acquired, 706; in bronchopneumonia, 694; congenital, 705
Athetosis, 954, 976
Athrepsia, 348
Athyrea, 901, 905
Atomization, 655
Atropine, doses of, in children, 470
Attenuants of cow's milk, 179
Aura, in grand mal, 926

B.

Babcock fat-tester, 124, 191
Bacillus, Brieger's, 177; of Eberth, 447; of Friedländer, 691; comma, of Koch, 505; Klebs-Lœffler, 459; lactis aërogenes, 87, 177, 182; of Pfeiffer, 476; of tetanus, 496; tubercle, 381
Bacteriology of cow's milk, 180
Balanitis, 870
Barley-jelly, preparation of, 239
  -water, preparation of, 239

Basophiles, 874, 878
Basophilic, 875
Bath, temperature of, 96
Bathing of infants, 96; in typhoid fever, 455
Bednar's aphthæ, 622
Beri-beri, 985
Bile at term, 38; in infancy and childhood, 83
Bile-ducts, congenital obliteration of the, 308
Biliary calculi, 837
Birth-palsies, 951
Bladder at term, 36; in childhood, 58; diseases of the, 865; reflex symptoms of the, 948; tuberculosis of the, 431
Blood, at term, 38; amount at birth, 876; average number of corpuscles at different ages, 878; color, 876; development of the, 56; diseases of the, 874; hæmoglobin, 876, 880; in abnormal conditions, 879; in chlorosis, 893; in infancy, 874; in leukæmia, 884; in normal conditions, 875; in pernicious anæmia, 890; in pseudo-leukæmia, 888; in secondary anæmia, 895; nomenclature of the, 874; percentages of various leucocytes in the, 879; reaction, 876; red corpuscles, 877, 880; specific gravity, 876, 880. (*Vide*, also, symptoms and diagnosis of the especial disease.)
Blood-corpuscles, description of different varieties of, 874; red, 877, 880. (*Vide*, also, Blood.)
Blood-vessels, diseases of the, 720
Bone, lesions of the, in cretinism, 901; in rhachitis, 329; in syphilis, 523, 528, 536
Bone-marrow, 81; at term, 37
Boracic acid, strength of, in local applications, 869
Bow-legs, 111
Brain, at term, 29; development of the, 47; in cerebro-spinal meningitis, 434; syphilis of the, 978, 980; tuberculosis of the, 406, 978
Branchial fistula, 296, 767
Brandy, dose of, 456
Breast-pump, 119
Breck's feeder for premature infants, 273
Bremer's blood-test in diabetes mellitus, 1006
Brieger's bacillus, 87, 177
Bright's disease, acute, 853; chronic, 857
Broadbent's sign, 765
Bromides, doses of, in epilepsy, 929
Bronchi, diseases of the, 665
Bronchial asthma, 673
Bronchiolitis exudativa of Curschmann, 673
Bronchitis, acute, 666; capillary, 697; chronic, 671; fibrinous, 672; unusual forms of, 669
Bronchocele, 899
Broncho-pneumonia, acute, 691; acute tubercular, 394; aspiration, 691; chronic, 704; definition of, 675; diagnosis, 700; etiology, 691; pathology, 692; physical signs in, 699; primary forms in, 691; prognosis, 700; secondary forms in, 691; symptoms, 696; terminations and complications in, 699; treatment, 701
Brooder for premature infants, 267
Brown atrophy of the heart, 734
Bruit de diable, 893
Buhl's disease in the new-born, 312
Bulbar paralysis, 985
Bursa pharyngea at term, 28

C.

Cachexia strumipriva, 901
Cæcum and ascending colon, relations of, 69
Calcareous degeneration of the heart, 734
Calculi, biliary, 837
Calomel, dose of, 796
Canal of Nuck, encysted hydrocele of, 306
Cancrum oris, 629
Capillary bronchitis, 697
Caput succedaneum, 286
Carcinoma of the brain, 978. (*Vide*, also, Tumors.)
Cardiac disease, *vide* Heart; dilatation, 732; hypertrophy, 731
Cascara sagrada, dose of, 805
Caseinogen, 178; definition of, 223
Castor oil, dose of, 796
Catalepsy, 935
Catarrhal fever, 476
Cephalhæmatoma, 287
Cephalodynia, 1001
Cereals in laboratory feeding, 202
Cerebral abscess, 949
meningitis, table of, diagnosis of, 415
palsies, infantile, 950
paralysis, 950
sinus, thrombosis of the, 975
syphilis, 978, 980
Cerebro-spinal fluid in cerebro-spinal meningitis, 439
Cerebro-spinal system, tuberculosis of, 405
Cervicodynia, 1001
Chenopodium, dose of the oil of, 832
Cheyne-Stokes respiration in acute broncho-pneumonia, 698
Chicken broth and jelly, preparation of, 244
Chicken-pox, 605
Chilblains, 365
Chloral, dose of, 574
Chloranæmia, 895
Chlorate of potassium, dose of, at different ages, 624
Chlorosis, 892
Cholera asiatica, 505
infantum, 502
Chorea, 916; congenital, 977; electrica, 924; habit, 924; hereditary or Huntington's, 924; post-hemiplegic, 954
Choreiform diseases, 924
Chondro dystrophia, 342
Chovstek's symptom, 943
Chronic fermental diarrhœa, 809
nervous diarrhœa, 798
Chyluria, 850
Cirrhosis of the liver, 838
Citrate of potash, dose of, 574
Cleft palate, 292
Clothing of infants, 98–102
Club-foot, 311
-hand, 311
Cocaine, doses of, 823; suppositories, 814

Colitis in measles, 593
Colles's law in hereditary syphilis, 521
Colon, dilatation of the, 810; irrigation of, the, 823
Colostrum milk, 122; chemistry of, 122; corpuscles, 122; period, 122; influence of, on the infant's weight, 76
Common carotid artery at term, 32
Compensation of the heart. (*Vide* the especial diseases of the heart.)
Concussion, 937
Condylomata in syphilis, 527
Congenital chorea, 977
  disease, definition of, 19. (*Vide*, also, Diseases of the new-born.)
  hydronephrosis, 865; stridor, 660
Congestion, chronic passive, of the kidney, 852
  of the liver, 835
Conglomerate tubercles, definition of, 384
Constipation, 798, 802; atonic, 803; spasmodic, 803
Convulsions, 911; clonic, 911; epileptic, 925; epileptiform, 925; general symptoms of, 912; of central origin, 912; of peripheral origin, 913; prognosis in, 914; reflex, 911, 913; tonic, 911; treatment of, 914
Conus arteriosus, stenosis of the, 725
Cor biloculare, 721; triloculare, 721
Cord, encysted hydrocele of the, 306; in cerebro-spinal meningitis, 434
Corrigan pulse in aortic insufficiency, 743
Corrosive sublimate, strength of, in local applications, 869
Coryza, acute, 640
Cow, the, adaptability of, for infant feeding, 169; Ayrshire, 168; Bretonne, 169; Brown Swiss, 169; care of the, 170; Devon, 168; Durham, or shorthorn, 168; Holstein-Friesian, 169; source of milk-supply, 168
Cow-pox. (*Vide* Vaccinia.)
Cow's milk. (*Vide* Milk of cows.)
Cows, tuberculin test of, 172
Craniotabes in hereditary syphilis, 524, 529; in rhachitis, 333
Cranium, 45; at term, 26. (*Vide*, also, Face and cranium.)
Cream, average analysis of 16 per cent., 223; definition of, 223.
Creolin, strength of, in local applications, 869
Cretinism, 901; endemic, 901; sporadic, 901
Cricoid cartilage, 42; distance of the, to sternum, 43
Croup, 662; false, 662; membranous, 662, 664
Culex, genus, in malaria, 482
Curschmann's bronchiolitis exudativa, 673; spirals, 673
Cyclic vomiting, 774
Cystitis, acute, 865; chronic, 865

D.

Dactylitis syphilitica, 524, 529
Degeneration, acute fatty, of the new-born, 312
Deglutition pneumonia, 691
Dental periods, 50
  reflexes, 943
Dentition, difficult, 634. (*Vide*, also, Teeth.)
Depressed sternum, 297
Dermatitis, 363; calorica, 365; exfoliativa neonatorum, 362; medicamentosa, 365; traumatica, 364; venenata, 364
Descending colon in infants, 70
Development, arrested psychical, 938: normal, 39
Dextro-cardia, 721
Diabetes insipidus, 1003; mellitus, 1004; pancreatic, 1005
Diabetic coma, 1006
Diacetic acid in diabetes mellitus, 1006
Diameters of thorax at term, 30
Diaphragm at term, 31; relations of, 53
Diarrhœa, 793; acute fermental, 805; acute nervous, 796; chronic fermental, 809. (*Vide*, also, symptoms of the especial disease.)
Diet in the regimen of lactation, 136. (*Vide*, also, Feeding, and the treatment of the especial disease.)
Difficult dentition, 634
Digitalis, doses of, 470
Dilatation, cardiac, 732
Dilutions of cream, with solutions of sugar, 231; with water, 231; with whey, 233; with whole milk, 231
Diphtheria, 459; antitoxin in, 468, 470; atypical infections of throat in, 464; complications and sequelæ, 466; diagnosis, 466; efflorescence in, 467; etiology, 460; incubation, 462; intubation in, 474; laryngeal, 465; malignant forms of, 464; nasal, 465; operative treatment, 474; pathology, 461; prognosis, 467; prophylaxis, 468; serum-therapy in, 470; symptoms, 462; tracheotomy in, 474; treatment, 469; variations of type in, 464
Diplegia, 953
Diplococcus intracellularis meningitidis, 432
Disseminated sclerosis, 987
Diuretin, dose of, 747
Drugs in disturbed lactation, 138
Ductless glands, diseases of the, 899
Ductus arteriosus, persistence of the, 725; post-natal changes in the, 55
Ductus venosus, post-natal changes in the, 55
Duodenal indigestion, acute (*vide* Intestinal indigestion), 797; chronic, 798
Duodenum at term, 34
Dura mater, 47
Dysentery (*vide*, also, Ileo-colitis), 820; chronic, 824; endemic, 501; tropical, 501
Dyspepsia, acute, 778; chronic, 779
Dystrophies, 990
Dyspnœa. (*Vide* symptoms of the especial disease.)

E.

Ear, the, 47; at term, 29; diseases of, 644; in cerebro-spinal meningitis, 439; in diphtheria, 465; in measles, 582, 591; in pneumonia, 682; in scarlet fever, 548, 570; in syphilis, 538; in variola, 599; reflex symptoms of, 944
Ear-drops, prescription for, 637
Ears, protrusion of the, 293
Eberth's bacillus, 447

Eclampsia infantum, 911
Eczema, 369; regional, 369; universal, 370
Emphysema, 706; compensatory, 706; in broncho-pneumonia, 696; interstitial, 706; obstructive, 706
Empyema, 713; in measles, 593; in pneumonia, 682
Emulsion in modified milk, 203
Encephalitis, acute, 984
Encephalocele, 290
Endocarditis, acute, 735; pathology, 736; septic or malignant, 735, 736, 738; simple or verrucous, 735, 736; treatment of, 739; ulcerative, 736
  chronic, 740; aortic insufficiency, 743; aortic stenosis, 744; diagnosis, 745; etiology, 740; general symptoms, 740; mitral insufficiency, 741; mitral stenosis, 741; pathology, 740; prognosis, 745; pulmonary insufficiency and stenosis, 745; treatment, 746; tricuspid insufficiency, 744; tricuspid stenosis, 744
Enemata, rectal, 830
Enteric fever, 447
Enuresis, 871; functional, 871; organic, 871
Eosinophiles, 875; percentage of, in the blood, 879
Eosinophilic, 875
Epidemic cerebro-spinal meningitis, 432; diagnosis, 440; etiology, 432; pathology, 433; prognosis, 440; symptoms, 436; treatment, 440; types of the disease, 435
Epidemic pemphigus infantilis, 362
Epididymitis, 870
Epiglottis, 43
Epilepsy, 925; grand mal, 925, 926; Jacksonian, 925; petit mal, 925, 926
Epileptic status, 927
Epileptiform convulsions, 925
Epiphyseal hyperæmia, 970
Epiphysitis, acute, 511
Epispadias, 308
Epistaxis, 644
Epsom salt, doses of, 833
Erb's juvenile form of progressive muscular atrophy, 993
Erysipelas, 497; ambulans, 497; migrans, 497; of the new-born, 498; of sucklings, 498
Erythema, 366; intertrigo, 367; multiforme, 366; neonatorum, 324; nodosum, 367; simplex, 366; urticatum, 367
Erythrocytes, 877, 880
Eustachian tubes, 48; at term, 28
Eustachian valve, post-natal change of the, 55
Examination and treatment, general principles of, auscultation, 250; history, 247; inspection, 248; lumbar puncture, 253; method of examining a child, 246; palpation, 248; percussion, 249; prophylaxis, 256; respiration, 248; Rontgen light, 253; temperature, 248; treatment in general, 255
Exanthemata, the, as a group, 542; differential diagnosis of, 612
Exercise in the regimen of lactation, 137
Exophthalmic goitre, 905
Extremities, diseases of the, in the new-born, 310; in rhachitis, 336; length of, in cretins, 902
Eye, at term, 29; in cerebro-spinal meningitis, 438; in measles, 592; in syphilis, 526, 538

F.

Face and cranium, at term, 26; development of, 45
Fæcal dejections, influence of fat on the color of the, 216
Fæces, incontinence of, 802
False asthma, 674
  croup, 662
Fat-free milk, definition of, 223
Fats in human milk, 124, 127, 128
Fatty degeneration, acute, in the new-born, 312
  of the heart, 734
Fatty infiltration of the heart, 734; of the liver, 836
Faucial tonsils at term, 28
Feeding, 160; after the twelfth month, 243; amounts of food in, 67, 68; amount at each, 188; apparatus, 186; by stomach-tube, 781; direct substitute, 160; directly from animals, 164; general principles of, 112; general rules for, during first year, 188; indirect substitute, 164; intervals of, 135, 188; intervals and number of, 136; maternal, *vide* Maternal feeding; mixed, 156; nipples in, 188; of average infants born at term, 214; of premature infants, 273; Ssnitkin's rule, 189. (*Vide*, also, Percentage feeding.)
Feet, 81; at term, 37; care of, in children, 102
Femoral hernia in the new-born, 305
Fibroid phthisis, 695
Filaria sanguinis hominis, 850
Fingers in the new-born, 310
Fissures, 813
Fistulæ, 814
Flat-foot, 81
Flexner's bacillus, 820
Fœtal circulation, 19
  rhachitis, 342
Fontanelles, at term, 25; development of, 45
Food, artificial, for infants, 240; malted, 241; management of, in early days of life, 135; source of, in indirect substitute feeding, 166
Foramen ovale, open, 723; post-natal changes in the, 55; premature closure of the, 723
Forced feeding, 781
Forchheimer's exanthem in rubella, 594
Foreign bodies in the larynx, 661; in the œsophagus, 768
Formulæ, general, for calculation of all percentage combinations, 235; for calculation of sugar percentage, 231; for cream and fat-free milk, 237; for cream and whey, 238; for cream and whole milk, 236
Fossa of Rosenmuller, 28

Fraenkel's pneumococcus, 675
Freckles, 376
Friedländer, bacillus of, 691
Friedreich's disease, 988
Frowlowsky's rule for gastric capacity, 59
Functions, development of, 82
Funnel chest in rhachitis, 334
Furunculosis, 358

G.

Gangrene of the lung, 707; in pneumonia, 682
Gastralgia, 777
Gastric catarrh, acute, 789; chronic, 792
Gastritis, acute, 789; catarrhalis acuta, 789; chronic catarrhal, 792; corrosiva acuta, 789, 791; pseudo-membranosa, 789, 791
Gastro-diaphane, 785
Gastro-enteric diseases, American Pediatric Society's classification of the, 769, 773; general bacteriology, 770; general diagnosis, 771; general etiology, 770; general symptomatology, 771; general treatment, 772; table of classification, 773
Gastro-enteric tract, tuberculosis of the, 399
Gavage, 780
Genitals, diseases of the, 865
German measles. (*Vide* Rubella.)
Giant urticaria, 379
Glioma, 978
Glossitis, 633
Glottis, œdema of, in measles, 593. (*Vide*, also, the especial disease.)
Glycosuria, 851; alimentary, 851; dietetic, 851; in scarlet fever, 573; temporary alimentary, 1005
Goitre, 899; exophthalmic, 905
Grand mal, 925, 926
Green sickness, 893
Growing pains, 970
Gum-lancing, 636
Gums at term, 27
Gyrospasm, 944

H.

Habit chorea, 924
Hæmatoblasts, 874
Hæmatoma of the sterno-cleido-mastoid, 295
Hæmaturia, 849; in chlorosis, 893; in pernicious anæmia, 890; in scorbutus, 344; in secondary anæmia, 895
Hæmoglobin, 875, 876, 880
Hæmoglobinæmia, 875
Hæmoglobinuria, 849, 875; paroxysmal, 849
    infectious, in the new-born, 314
Hæmolysis, 875
Hæmopericardium, 754
Hæmophilia, 997
Hands at term, 37
Hard palate, 29, 48
Harelip, 291
Harrison's groove in rhachitis, 334
Headaches, 939
Head, anencephalic, 290; at term, 25; development of the, 43; diseases of the, in the new-born, 286; in rhachitis, 333; macrocephalic, *vide* Hydrocephalus; microcephalic, 932
Hearing at term, 38
Heart, at term, 32; diseases of the, 719; in scarlet fever, 554; post-natal changes in the, 54; reflex symptoms of the, 948
    acquired diseases of the, 730; amyloid degeneration, 734; brown atrophy, 734; calcareous degeneration, 734; dilatation, 732; endocarditis, 735; fatty degeneration, 734; fatty infiltration, 735; hyaline transformation, 734; hypertrophy, 731; myocarditis, 733
    congenital diseases of the, 720; defect of the ventricular septum, 724; differential diagnosis of the, 726; general diagnosis, 723; general symptoms, 721; general treatment, 730; lesions of the aortic valve in, 726; lesions of the mitral valve in, 726; open foramen ovale in, 723; persistence of the ductus arteriosus in, 725; pulmonary atresia in, 725; pulmonary stenosis in, 724; transposition of the large arteries in, 726; tricuspid insufficiency in, 726; tricuspid stenosis in, 726; stenosis of the conus arteriosus in, 725
    functional diseases of the, 748
    murmurs. (*Vide* lesions of the especial valves.)
Heat-stroke, 935
Height, at term, 36, 73; in infancy and childhood, 73
Hemiplegia, 953
Hemitaxia, 954
Hemorrhage in early life, 317; in scorbutus, 344; in syphilis, 526; in typhoid fever, 448, 450. (*Vide*, also, symptoms of the especial disease.)
Hemorrhagic diathesis, 997
Hemorrhagic disease of the new-born, 317
Hemorrhoids, 814
Henoch's purpura, 999
Hepatitis, interstitial, 838; atrophic and hypertrophic, 838; suppurative, 836
Hepatization, red and gray, 676
Hereditary ataxia, 988
    chorea, 924
    spastic paralysis, 989
    syphilis. (*Vide* Syphilis.)
Hernia, 813; congenital, funicular, and infantile, 304; inguinal, 304
Herpes zoster, 374
Hip, congenital dislocation of the, 311; tuberculosis of the, 429
Hives, 367
Home modification of milk, 217; apparatus for, 224; definition of terms in, 223; difficulties and dangers of, 217; final remarks on, 239; materials for, 223; method of obtaining cream and separated milk for, 225; prescriptions for, 226; table for calculation of, 229. (*Vide*, also, Formulæ.)
Human milk. (*Vide* Milk, human.)
Huntington's chorea, 924
Hyaline transformation of the heart, 734
Hydatids of the liver, 837
Hydrocele, 305; congenital, infantile, and funicular, 305; in the new-born, 305

Kidneys, at term, 33; in cerebro-spinal meningitis, 435; in childhood, 57; enlargement of the, 866; in measles, 592; in scarlet fever, 553, 572; diseases of the, 844
  acquired diseases of the, *vide*, also, Nephritis; active hyperæmia, 851; acute pyelitis and pyelonephritis, 862; albuminuria of adolescence, 848; amyloid infiltration, 859; anuria, 847; chronic passive congestion, 852; general etiology of, 844; general pathology of, 844; general symptomatology of, 846; hæmaturia, 849; hæmoglobinuria, 849; malignant growths, 866; passive hyperæmia, 852; physiological albuminuria, 847; tuberculosis, 431
  congenital diseases of the, 844
Klebs-Loeffler bacillus, 459
Knee, congenital dislocation of the, 311; tuberculosis of the, 429
Knock-knee, 111
Koplik's gastro-diaphane, 785
  spots in measles, 583; in rubella, 594
Kumyss, 241
Kyphosis in rhachitis, 336

L.

Laboratory modification of milk, practical limits of, 199
Lachrymal glands at term, 38; development of, 82
Lactalbumin, 178; definition of, 223
Lactation, disturbed, 138; analyses of human milk in, 143; general principles for the management of, 140, 141; menstruation in, 139
  prolonged, 156
  regimen of, 136
Lacto-globulin, 178
Lagophthalmia, 969
La grippe, 476
Landouzy-Déjerine type of progressive muscular dystrophy, 993
Landry's paralysis, 986
Large intestines at term, 36; development of, 69
Laryngismus stridulus, 660
Laryngitis, 662; acute catarrhal, 662; chronic, 662; membranous, 465, 593, 664
Laryngospasmus, 660, 944
Larynx, 43; at term, 25; diseases of the, 660; foreign bodies in the, 661; new growths, 660; œdema of, 662; pseudo-membranous, 664; reflex symptoms of the, 944
Lavage, 780
Leg type of progressive muscular atrophy, 991
Lentigo, 376
Leptomeningitis, 981
Letter S curve, 710
Leucocytes, 874, 881; in children, 877
Leucocytosis, 875; in children, 881; in secondary anæmia, 895. (*Vide*, also, symptoms and diagnosis of the especial disease.)
Leukæmia, 882; lymphatic, 883; splenic myelogenous, 882
Lichen planus, 377
Lingua geographica, 633
Lipoma of the brain, 978
Liver, abscess of the, 836; acute yellow atrophy of the, 835; anatomy, 834; at term, 33; amyloid, 837; in cerebro-spinal meningitis, 435; in childhood, 57; cirrhosis, 838; congestion, 835; diseases of the, 834; fatty infiltration, 836; hydatids, 837; icterus, 835; new growths, 837; in syphilis, 522; tuberculosis of, 431
Lobular pneumonia, 691. (*Vide*, also, Broncho-pneumonia.)
Locomotor ataxia, 989
Lordosis in rhachitis, 336
Lumbago, 1001
Lumbar puncture, 253
Lungs, the, abscess of, 707; acute miliary tuberculosis of, 393; at term, 32; chronic localized tuberculosis of, 395; chronic diffuse tuberculosis of, 395; development of, 56; diseases of, 665; gangrene of, 707; in cerebro-spinal meningitis, 434, 438; reflex symptoms of, 946; syphilis of, 523; tuberculosis of, 393
Lupus, 432
Lymphangioma cavernosum, 634
Lymphatic leukæmia, 883
  system at term, 39; development of, in early life, 83
Lymphatism, 1000
Lymph-nodes, diseases of the, 897; in cerebro-spinal meningitis, 435
  cervical, in measles, 592; in scarlet fever, 549, 569; in syphilis, 525; in tuberculosis, *vide* Tuberculosis of the lymph-nodes
Lymphocytes, 878; large, small, mononuclear, and transitional, 874
Lymphoid tissue at term, 28
Lymph vessels of the pharynx at term, 27

M.

Macrocytes, 874
Macroglossia, 634
Malaria, 481; chronic, 488; diagnosis, 488; etiology, 481; genus anopheles in, 481; genus culex in, 481; intermittent, 485; pathology, 485; paroxysms in, 486; prognosis in, 488; prophylaxis in, 489; remittent, 485; symptoms, 485; subacute forms, 488; treatment, 489
Male fern, oleoresin of, dose of, 832
Malformations of intestines, 795
Malignant endocarditis, 736, 738; septic type, 739; typhoidal type, 739
Malignant growths. (*Vide* New growths and tumors.)
Malposition of the intestines, 795
Malted foods, 241
Mammary gland, 114
Marasmus, 348
Marmorek's antistreptococcus serum in scarlet fever, 562
Mastitis, 118; in the new-born, 296
Mastoid antrum at term, 29
Masturbation, 873
Maternal feeding, 116; contraindication to, 117
Maternal impressions, 285
Matzoon, 241
Maxillary bones, 48

Measles, 579; complications and sequelæ of, 590; contagium in, 580; desquamation, 584, 589; diagnosis, 584; efflorescence, 584, 588; etiology, 579; incubation, 582, 588; hemorrhagic or malignant form, 588; pathology, 581; prodromata, 582, 588; prognosis, 585; recurrent, 589; relapses, 589; symptoms, 582; treatment, 585; variations in type, 587
Meckel's diverticulum, 303
Meconium, 39
Megaloblasts, 874
Megalocytes, 874
Melæna neonatorum, 320
Melanoderma lenticularis progressiva, 376
Meningitis, 981; basilar, 982; cerebral, table of diagnosis, 415; epidemic cerebrospinal, 432; in pneumonia, 684; serosa, 971; simple acute, 982; tubercular, 406; tubercular, infantile form, 410
Meningocele, 289
Meningo-myelitis, 958
  -myelocele, 298
Menstruation in disturbed lactation, 139
Mental development in syphilis, 537
Mental impressions, development of, 82
Mercurials, use of, in syphilis, 531
Microblasts, 874
Microcephalus, 932
Micrococcus lanceolatus, 676
Microcytes, 874
Microglossia, 634
Microsporon furfur, 360
Migraine, 940
Miliary tubercles, definition of, 384
Miliary tuberculosis, 387. (*Vide* Tuberculosis.)
Milk, 120; care of, 172; formation of, 120; home modification of, 217 (*vide*, also, Home modification); nervous disturbances affecting the, 121; peptonization of, 202
  human, 124; analyses in disturbed lactation, 143; average analyses of, 127 (*vide*, also, the analyses in the illustrative cases of breast and substitute feedings, 143–155 and 210–213); bacteriological examination of, 134; clinical examination of, 124; clinical significance of the chemistry of, 127; fats, 124, 128; lactose or milk-sugar, 126, 129; microscopic examination, 126; mineral matter, 126, 129; proteids, 125, 129, 200; specific gravity, 124, 128; water, 128; variations, 132
  -laboratories, 184, 189; apparatus for transportation of modified milk, 193; Babcock fat-tester, 191; milk-room, 190; modifying-room, 191; separating-room, 191; separator, 191; sterilization of the milk, 196; still, 191; ventilator, 191; wash-room, 197
  of cows, as compared with woman's milk, 180; attenuants, 179; average analysis, 173; bacteriology, 180; fats, 175; lactose or milk-sugar, 176; milk plasma, 176; mineral matter, 178; proteids, 177, 200; reaction, 174; specific gravity, 175
  of magnesia, dose of, 805
Mirror writing, 932
Mitral insufficiency, 741
  orifice, lesions of, in congenital disease, 726
  stenosis, 741
Modification of milk, materials for, 195; practical limits of, in the milk-laboratories, 199. (*Vide*, also, Formulæ, Home modification, and Feeding.)
Modified milk, the emulsion in, 203
Molluscum contagiosum, 358
Monoplegia, 953
Morbus maculosus Werlhofii, 999
Morphine, doses of, 505, 824
Mouth at term, 29; care of the, 107; in syphilis, 527
  diseases of the, 615; nomenclature, 615; table of classification of, 616
  -wash, 653
Mucous polypi of the nose, 643
Multiple neuritis, 985
Mumps, 613; submaxillary, 614
Murmurs, heart, congenital, 727; functional, 749; organic, 749. (*Vide*, also, the especial valvular lesion.)
Muscular atrophy, progressive central, 989; progressive neural, 990, 991
  dystrophies, atrophic form, 995; classification of the, 993; Erb's juvenile form, 993, 995; Landouzy-Déjerine type, 993, 995; pseudo-hypertrophic form, 994
  rheumatism, 1001
  tremors in exophthalmic goitre, 906
Mutton broth, preparation of, 245
Myalgia, 1001
Mycelium, 626, 628, 629
Myelitis, acute, 958
Myelocytes, 875, 878; eosinophilic, 875; neutrophilic, 875
Myocarditis, interstitial, 733; parenchymatous, 733
Myoclonia, 925
Myomalacia, 734
Myopathies, 990; primary, 993
Myotonia congenita, 924
Myotonic reaction, 924
Myxœdema, 901; acquired, 901; congenital, 901; operative, 901
Myxoma of the brain, 978

N.

Nævus, 325
Nails in syphilis, 526, 538
Napkins, 39, 84, 87, 793
Nasal irrigation, 473
Naso-pharynx, 47; at term, 27; diseases of the, 645
Natiform skull in late hereditary syphilis, 537
Neck, at term, 24; development of the, 42
  diseases of the, in the new-born, 295
Nephritis, acute, 853; acute diffuse, 853; catarrhal, 852; chronic interstitial, 857; chronic parenchymatous, 855; subacute glomerular, 855. (*Vide*, also, Kidney.)
  pathology of acute degenerative, acute glomerular, acute hemorrhagic, acute interstitial, 549–553, 853

Nerves and nerve ganglia in cerebro-spinal meningitis, 434
Nervous disturbances affecting the milk, 121
  system, diseases of the, 910
    in cerebro-spinal meningitis, 433
Nettle-rash, 367
Neuralgia, 970
Neuritis, multiple, 985
Neutrophiles, 875
Neutrophilic, 875
New-born, the, acute fatty degeneration of, 312; asphyxia in, 312
    diseases of, 282; erysipelas of, 498; extremities in, 310; general diagnosis, 285; general diseases, 312; general etiology, 283; general pathology, 283; general prognosis, 285; general symptoms, 284; head in, 286; hemorrhage in the, 317; hemorrhagic disease of, 317; infectious hæmoglobinæmia of, 314; infectious hæmoglobinuria of, 314; inheritance in, 282; malformation in, 282; maternal impressions as a factor in, 285; neck in, 295; paralysis of, 968; syphilis of, 521; traumatism in, 282; trunk in, 296 .
New growths of the intestines, 814; of the larynx, 660; of the liver, 837; of the stomach, 783, 789. (*Vide*, also, Tumors.)
Nipples, 118, 188
Nitrate of silver, strength of, in local applications, 869
Nitroglycerin, doses of, in children, 470
Noma, 629; in measles, 593
Normal development, 39
  infant, the, at term, 18
  maternal conditions, 116
  salt solution, dose for subcutaneous injection of, 504
Normoblasts, 874
Nose, diseases of the, 640
Nursery, the, 93; bed, 94; closets and drawers, 94; curtains, 94; draughts, 95; floor, 93; furniture, 94; heating and ventilation, 95; papers and carpet in, 93; picture mouldings, 93; pillow and mattress, 94; rugs, 93; scales, 95; sun and windows, 93; toys, 94; walls and ceilings, 93
Nursery-maids, 107
Nursing, 117; irregularity in, 136
Nutrition, diseases of, 326; relation of weight to, 73–81
Nutritive period, first, 116; second, 241; third, 245
Nux vomica, dose of, 805
Nystagmus, 944, 988

O.

Oat-jelly, preparation of, 239
Obstetrical paralysis, 968
O'Dwyer's tubes in intubation, 476
Œdema of the larynx, 662; neonatorum, 323. (*Vide*, also, symptoms of the especial disease.)
Œsophagitis, 767
Œsophagus, congenital dilatation of the, 769; diseases of the, 767; foreign bodies in the, 768
Oïdium albicans, 626
Oligocythemia, 875
Omodynia, 1001
Onychia, 526, 538
Ophthalmia neonatorum, 294; catarrhal, 294; purulent, 294
Opium, doses of the tincture of, 823
Orchitis, 870
Ossification of the sternum, 30; of the thorax, 51
Osteomalacia, 343
Osteomyelitis, acute infectious, 511
Osteoperichondritis in hereditary syphilis, 524, 528
Osteoperiostitis in hereditary syphilis, 523
Otitis media. (*Vide* Ear.)
Oxyphiles, 875
Oxyuris vermicularis, 829
Ozæna, 643

P.

Pachymeningitis, 981
Palsies, birth, 951; extra-uterine, 951; infantile cerebral, 950
Paludism. (*Vide* Malaria.)
Pancreas, the, at term, 38; in childhood, 57; development of the function of, 83; diseases of the, 840; tuberculosis of, 430; in syphilis, 523
Pancreatic diabetes, 1005
Paralysis, bulbar, 985; caused by caries of the spine, 967; cerebral, 950; hereditary spastic, 989; in cerebro-spinal meningitis, 437; infantile spinal, 958; in measles, 593; in tuberculosis of the spine, 429; Landry's, 986; obstetrical, 968
Paramyoclonus multiplex, 925
Paramyotonia, congenital, 925
Paraplegia, 953
Parasites. (*Vide* Animal parasites.)
Parotitis, 613
Paroxysmal gasping, 946
Parrot's disease, 528
Pavor nocturnus, 941
Pectus carinatum, 334
Pediculosis, 357
Pediculus capitis, 357
Peliosis rheumatica, 998
Pelletierine, dose of, 832
Pelvis at term, 36
Pemphigus, 361; neonatorum, 361; purulent, 362; syphilitic, 525
Peptonized milk, 241
Percentage combinations for premature infants, 274
  feeding, illustrative cases in, 206; principles of prescription writing in, 197; use of whey in, 200, 201; whey-cream mixtures in, 201
Perforation in typhoid fever, 448, 450
Pericarditis, acute, 754; diagnosis, 759; dry, 755; etiology, 754; in measles, 593; plastic, 755; paracentesis in, 762; pathology, 754; physical signs, 755; in pneumonia, 682; treatment 762; with exudation, 755

Q.

Quartan parasite, 483, 484
Quinine, dose of, in malaria, 489

R.

Rachischisis, 301
Ranula, 293
Reaction of degeneration, 962
Rectum, imperforate, 307; malformations about the, 307; reflex symptoms of the, 949
Red blood-corpuscles, 877, 880
Reflex cough, 947
Reflex symptoms, of the bladder, 948; of the ear, 944; of the heart, 948; of the larynx, 944; of the lung, 946; of the rectum, 949; of the stomach 948; of the vagina, 948
Retarded speech, 938
Retropharyngeal abscess, 656
Respirations at term, 30, 36, 72; in infancy, 53; in infancy and childhood, 72
Retinitis in scarlet fever, 573
Rhachitis, 326; acute, 333; congenital, 342; diagnosis, 338; fœtal, 342; late, 327; of adolescence, 327; pathology, 329; prognosis, 340; rosary, 334; symptoms, 332; treatment, 341
Rhagades in hereditary syphilis, 528
Rheumatic fever, 514; subcutaneous fibrous nodules in, 519
Rheumatism, acute articular, 514; chronic, 1003; muscular, 1001
Rhinitis, 640; acute, 640; atrophic, 643; hypertrophic, 642; purulent, 642
Rhus toxicodendron, 364
    venenata, 364
Rickets (*vide* Rhachitis), 326
Ringworm, 359
Ritter's disease, 362
Röntgen light, 253
Rosary in rhachitis, 334
Rose-spots in typhoid fever, 450; typhoid bacilli in the, 452
Rotch's experimental work on pericarditis, 757
Rotheln. (*Vide* Rubella.)
Rubeola. (*Vide* Measles.)
Rubella, 593
Rupia, 526

S.

Salicylates, dose of the, 517
Salivary glands at term, 38; development of, 83
Salt solution, normal, dose for subcutaneous injection of, 504
Santonin, dose of, 830
Sarcoma of the brain, 978
Scabies, 355
Scanning speech, 987
Scapulodynia, 1001
Scarlatina, *vide* Scarlet fever; sine eruptione, 566
Scarlet fever, 543; contagium, 545; etiology, 543; incubation, 554; pathology, 546; pseudo-relapses in, 365; recrudescence, 566; reinfections, 365; relapses, 365; sine eruptione, 566; variations in the type, 554
Scarlet fever, benign form of, 554; complications of 568; desquamation, 555; diagnosis, 557; efflorescence, 555; isolation and disinfection, 562; prodromata, 554; prognosis, 557; prophylaxis, 558; symptoms, 554; treatment, 559; variations in the type, 565
    malignant form of, 578
Schönlein's disease, 998
School, influence of, on the child, 108
Sciatica, 970
Sclerema neonatorum, 322
Scleroderma, 379
Sclerosis, insular or disseminated. 987
Scoliosis in rhachitis, 336
Scorbutus, 344
Scrofula, 390, 432
Scrofuloderma, 432
Scurvy, 344
Sebaceous glands at term, 38
Seborrhœa capitis of infants, 359
Septic endocarditis, 739
Senna, dose of, 832
Separated milk, definition of, 223
Shoes for children, 102
Sight, function of, at term, 37
Sigmoid flexure in infancy and childhood, 70
Simulated diseases, 935
Skeletons, infantile, 87
Skin, 81; at term, 21; diseases of the, 355; in cerebro-spinal meningitis, 435, 438; in measles, 581; in scarlet fever, 546; in syphilis, 525; in tuberculosis, 432
Skoda's resonance, 710
Sleep, amount of, for infants and children, 106
Small intestines at term, 36; development of, 68
Small-pox, 595; modified, 599. (*Vide* Varioloid.)
Smell, function of, at term, 38
Snuffles in hereditary syphilis, 524
Soft-palate at term, 29
Spasmus nutans, 944
Spastic paralysis, hereditary, 989
Specific infectious diseases, 381
Speech, retarded, 938
Spigelia, dose of, 832
Spina bifida, 298
Spinal meningocele, 288
Spinal paralysis, infantile, 958; temporary, 961
Spine, the, 39; at term, 22; curves, 41; flexibility, 40; length, 40; surface anatomy, 41; in rhachitis, 338; tuberculosis of, 429
Spleen, diseases of the, 840; in childhood, 57; in cerebro-spinal meningitis, 435; in rhachitis, 331; in syphilis, 523; in typhoid, 450; in tuberculosis, 431
Splenic myelogenous leukæmia, 882
Spondylitis, tubercular, 407
Spray for throat, 655
Status lymphaticus, 1000
Sternum, the, at term, 30
Stomach, the, at term, 34; capacity, 58; development, 58; developmental diseases, 774; malformations, 774; malpositions, 774

- skin, 432; spine, 429; spleen, 431; testicle, 431; thymus gland, 430; thyroid gland, 430; trachea, 393
- Tuberculosis, acute miliary, 387; of the lung, 395; simulating infantile atrophy, 388; simulating typhoid fever, 387
  - general, 386; chronic, 389
  - of the lymph-nodes, 389; bronchial, 392; cervical, 390; mesenteric, 392
  - relation of bovine, to human tuberculosis, 381
- Tubular indigestion, 798
- Tumor albus, 429
- Tumors, intracranial, 978
- Turpentine stupe, 456
- Typhoidal ileo-colitis, 447
- Typhoid bacillus in cerebro-spinal fluid, 450
- Typhoid fever, 447; complications and sequelæ, 451; diagnosis, 451; diazo-reaction in, 450; differential diagnosis, 452; etiology, 447; incubation, 449; in infancy and early childhood, 447; in the fœtus, 447; pathology, 448; prognosis, 454; symptoms, 449; treatment, 454; Widal reaction in, 450
- Typhus fever, 459

U.

- Ulcers of the stomach, 783, 786
- Umbilical arteries, post-natal changes in the, 55
  - cord, 21, 39, 82
  - hernia, congenital, into the cord, 302; in the new-born, 302
  - vein, post-natal changes in the, 55
- Umbilicus, fungus of the, 302; polypus of the, 302
- Unclassified diseases, 997
- Undescended testicle, 306
- Uræmia in acute diffuse nephritis, 855; in chronic interstitial nephritis, 858; treatment of, 574. (*Vide*, also, symptoms of the especial disease.)
- Uric acid infarction at term, 34
- Urine, at term, 39, 85; diazo-reaction in the, 452; in acute cystitis, 867; in active hyperæmia, 851; in acute pyelitis and pyelonephritis, 863; in acute diffuse nephritis, 854; in amyloid infiltration, 859; in chronic cystitis, 867; in chronic interstitial nephritis, 858; in chronic parenchymatous nephritis, 856; in chronic pyelitis, 864; in chyluria, 850; in diabetes insipidus, 1004; in diabetes mellitus, 1006; in glycosuria, 851; in hæmaturia, 849; in hæmaglobinuria, 849; in hydronephrosis, 865; incontinence of, 871; in infancy and childhood, 85; in passive hyperæmia, 852; in physiological albuminuria, 847; in scarlet fever, 556; in subacute glomerular nephritis, 856. (*Vide*, also, symptoms of the especial disease.)
- Urotropin, dose of, 867
- Urticaria, 367
- Uterus at term, 36
- Uvula at term, 29
  - elongation of the, 655

V.

- Vaccinia, 601
- Vaccination, 601
- Vagina, occlusion of the, 307; reflex symptoms, 948
- Varicella, 605; complications, 608; diagnosis, 608; etiology, 605; gangrenous, 606, 608; pathology, 606; prognosis, 609; symptoms, 606; treatment, 609
- Variola, 595; complications, 599; confluent form, 598; diagnosis, 600; discrete form, 597; etiology, 595; hemorrhagic form, 598; incubation, 597; modified form, 598; pathology, 595; prognosis, 601; symptoms, 597; treatment, 601
- Varioloid, 599
- Veins at term, 32
- Ventricular septum, defect of the, 724
- Vermiform appendix, 815 (*vide*, also, Appendicitis); in infancy and childhood, 70
- Vernix caseosa, 21
- Verrucæ, 375
- Verrucous endocarditis, 721
- Vertigo, 940
- Vitality at term, 37
- Voice, function of, at term, 37; development of, 82
- Volvulus, 811
- Vomiting, acute nervous, 774; cyclic or persistent, 774. (*Vide*, also, symptoms of the especial disease.)
- Von Jaksch's pseudo-leukæmia of infancy, 887
- Vulvo vaginitis, 868

W.

- Warts, 375
- Waxy rigidity, 935
- Weaning, 157
- Weight, at term, 36; development of, 55; general figures of, 79; of infants and children, 73; of premature infants, 265; relation of nutrition to, 73–81
- Westcott's general formulæ, 235; tables of dilutions of creams, 232
- Wet-nurses, 162; diet of, 163; in cases of syphilis, 531
- Wheat-jelly, 240
- Wheat-water, 240
- Whey, definition of, 223; average analysis of, 223; formulæ for cream and, 238; in percentage feeding, 200; preparation of, 235; with dilutions of cream, 233
- Whey-cream mixtures, prescriptions for, 201
- Whey-proteids. (*Vide* Lactalbumin.)
- White blood-corpuscles, 881
- Whiskey, doses of, 456
- Whole milk, definition of, 223; average analysis of, 223
- Whooping-cough, 505
- Widal reaction, 451
- Winckel's disease, 314

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on
or before the date last stamped below.

P45 Rotch, T.M. 66439
R842 Pediatrics.
1901

NAME	DATE DUE